University of Alberta

MECHANICAL STENOSIS OF THE MAXILLARY ARTERY IN THE INFRATEMPORAL FOSSA: AN ETIOLOGY OF CRANIOMANDIBULAR PAIN AND VASCUALR HEADACHE

by

Danny Robert Kolotyluk ©

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Masters of Science

Department of Radiology and Diagnostic Imaging

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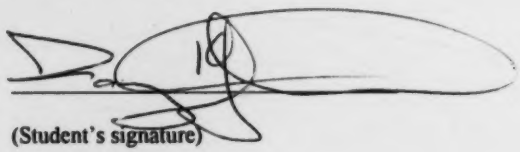

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ABSTRACT

The role of hypertrophy due to parafunctional teeth clenching of the two bellies of the lateral pterygoid muscles are assessed in the etiology of craniomandibular pain and vascular headache. A sample of 46 volunteers who suffer from facial pain and headache were examined with the use of magnetic resonance imaging and doppler ultrasound to assess the effect of the muscle bodies upon the maxillary artery during clenching. There was noted signs of vascular stenosis (flow disturbance including turbulence and bruits) due to mechanical compression within the infratemporal fossa. The effect of this vascular flow disturbance resulted in ischemia to the gasserian ganglion and the dura. Mechanical stimulation of the maxillary artery and its tributaries vessel wall also contributed to pain development. The gasserian ganglion and the trigeminal nucleus has been implicated in the genesis of vascular headache.

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Table of Contents

CHAPTER		Page
1.		2.
INTRODUCTION		2.
1.1	The Problem	3.
1.2	Proposal	5.
1.3	Objectives	5.
2.		7.
REVIEW OF THE LITERATURE		7.
2.1	Myofascial Pain and Mandibular Clenching	8.
2.2	Mandibular Clenching and Masticatory Muscles	9.
2.3	Electromyography of the Lateral Pterygoid Muscles	12.
2.4	Maxillary Artery and the Lateral Pterygoid Muscle	16.
2.5	Pain Genesis From the Maxillary Artery	17.
2.6	Pain Genesis and the Trigeminal Nerve	18.

3. 22.

MATERIALS AND METHODS 22.

3.1 Population Sample 23.

3.2 MRI 24.

3.3 MRI Technique 25.

3.4 Ultrasound Imaging and Doppler 26.

3.5 Doppler Imaging 28.

4. 32.

RESULTS and DISCUSSION 32.

4.1 Population 33.

4.2 MRI Results 36.

4.3 Ultrasound Imaging 44.

4.3a Ultrasound Measurement of Maxillary Artery 44.

4.3b Color Flow Doppler Imaging 48.

4.4 Spectral Doppler Measurements 52.

4.4a Typical Spectral Waveform Change from Rest to Clench to Rest 52.

4.4b Severe Stenosis 58.

4.4c Significant Peak Velocity Reduction and Spectral Disturbance 63.

4.4d Flow disturbance and Occlusion 65.

5. 84.

CONCLUSIONS 85.

6. 92.

FUTURE WORK 93.

BIBLIOGRAPHY 96.

Appendix I 117.

ANATOMY 117.

Local anatomy of the maxillary artery 118.

Appendix II 124.

Craniomandibular index form 125.

Appendix III 126.

III.1 Spectral analysis 127.

III.2 Waveform 128.

III.3 Arterial stenosis 130.

Appendix IV 135.

Ethics Approval 136.

List of Tables

Table		Page
1.	CMI Pain Indices	35.
2.	Normal Lateral Pterygoid Belly	41.
3.	Symptomatic Lateral Pterygoid Belly	43.
4.	Peak Artery Velocities Symptomatic	68.
5.	Peak Artery Velocities Normal	69.

List of Figures

Figure		Page
1.	Axial MRI	36.
2.	Coronal MRI	38.
3.	Sagittal MRI	39.
4.	Sagittal MRI of TMJ	40.
5.	Lumen 0.3 cm	45.
6.	Clench lumen 0.15 cm	46.
7.	Relax lumen 0.22 cm	47.
8.	Color flow doppler, jaws at rest	49.
9.	Color flow doppler, jaws clenched	50.
10.	Color flow doppler, jaws relaxed	51.
11.	Rest velocity 65.4 cm/sec	53.
12.	15 sec clench velocity 85.4 cm/sec	55.
13.	30 sec clench velocity 77.1 cm/sec	56.
14.	45 sec clench velocity 48.7 cm/sec	57.
15.	60 sec clench velocity 32.5 cm/sec	58.
16.	Rest flow 88.6 cm/sec	59.
17.	Clench flow velocity 43.4 cm/sec	60.
18.	Bruit formation	61.
19.	Return rest flow	62.
20.	Rest flow velocity 70.5 cm/sec	63.
21.	Clench velocity 25.5 cm/sec	64.
22.	Rest flow velocity 43.9 cm/sec	65.
23.	Disrupted flow velocity 21.6 cm/sec	66.

List of Figures

Figure		Page
24.	Anatomy of external carotid	119.

List of Graphs

Graph		Page
1.	Age/Pain indices	70.
2.	Age/Left muscle ratio	71.
3.	Pain/Left muscle ratio	73.
4.	Pain/Right muscle ratio	74.
5.	Age/Left rest peak velocity	76.
6.	Age/Right rest peak velocity	77.
7.	Age/Left clench peak velocity	78.
8.	Age/Right clench peak velocity	79.

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CHAPTER 1

INTRODUCTION

1.1 The Problem

Pain in the region of the infra-temporal fossa in the area of the temporomandibular joint and headache are difficult processes to decipher. Investigation into myofascial pain dysfunction has offered no well defined etiology for its existence. The process of pain in this area has produced diagnostic dilemmas. Many theories have been advanced regarding genesis of pain but for the most part there still does not exist agreement amongst practitioners of a diagnostic criteria for this complex regional pain syndrome (99, 100, 101, 102, 103, 104, 105,106, 107). These problems are two-fold. First, clinicians have not come to agreement on a diagnosis, and lacking this, they cannot agree on treatment protocols. A major stumbling block to this diagnostic confusion may be partly the prejudices and preconceived notions of the clinicians. Laszlo Schwartz 1955 (11) stated " The clinician's concept of etiology will influence what he looks for in his examination as well as what he tries to manipulate in therapy". Pain in the face and jaws was often related to functional disorders of the masticatory system including restriction of mandibular movement. Unfortunately, pain at rest mixes this functional disorder into a group of heterogeneous and often poorly defined diseases associated with headache and facial pain.

Localized tenderness to digital palpation of the muscles of mastication is a major finding in clinical and epidemiological studies of functional disorders of the masticatory system. (2,3,5,6, 7, 10, 11, 12,14, 16, 17,18) The perceived tenderness has been taken as a sign of muscle hyperactivity although there is little evidence to support this assumption.

Due to their specific location, pain within the muscles of mastication have been experienced as headache or facial pain. This pain development was thought to have developed through sustained contracture of masticatory muscles leading to fatigue and ischemia (12, 13, 14, 15, 16, 17). The theory was sustained and rhythmic contractions of these muscles at a high rate will result in localized pain due to obstruction of arterial flow and venous outflow caused by the contraction itself. There was thought compression of both arterial and venous return was generating localized ischemia.

1.2 Proposal

I am postulating an etiology of this diagnostically difficult painful condition that suggests its roots are more centrally located. Transient compression by muscle

hypertrophy of the first and second segment of the maxillary artery as it progresses through the infratemporal fossa suggests a more comprehensive explanation of the complex pain problems and headache experienced by the patient. The arterial flow for the muscles of mastication, the temporomandibular joints and the mandible arise from the external carotid system of which the maxillary artery is one of two terminal branches. The compression of the maxillary artery is postulated due to parafunctional hypertrophy of the upper and lower bellies of the lateral pterygoid muscle as the maxillary artery traverses between the two bellies(see Anatomy Appendix I).

1.3 Objectives

1. To demonstrate anatomically, using magnetic resonance imaging, the relationship of the lateral pterygoid muscle bodies and the maxillary artery.
2. To image the maxillary artery through the coronoid notch window using color flow doppler ultrasound and to demonstrate signs of compression upon the artery during clenching of the teeth of the volunteer and then during relaxation.
3. To record and analyze the spectral trace from

doppler ultrasound to demonstrate signs of flow disturbance including development of turbulence during clenching of the teeth.

The selection of the volunteer group included only those persons who suffer from pain. This precluded any age matched groups and selection of normals from the general population. Demonstration of changes within the lateral pterygoid muscle bodies and their effect upon the intraluminal diameter of the maxillary artery when the lateral pterygoid muscles were contracting was of prime interest.

CHAPTER 2.

REVIEW OF THE LITERATURE

2.1 Myofascial Pain and Mandibular Clenching

Costen, in 1935 described facial pain, earache, and headache that he felt was caused by dysfunction of the craniomandibular articulation (5). He termed this malady Costen's Syndrome, and in spite of a number of generations of health practitioners renaming, redefining and re-examining this condition, there exists within the medical system a limitation of knowledge dispersion such that the condition often is still referred to as Costen's Syndrome.

The portion of this syndrome of interest to this investigation is parafunctional clenching of the maxillary and mandibular teeth. Clenching is considered to be parafunctional if it involves long-lasting tooth contacts when the subject is neither chewing nor swallowing(6).

If the dental clenching is performed primarily with the mandible in a protrusive posture the masseter and pterygoid muscles are most active. This hyperactivity of the masticatory muscles results in fatigue and pain. The resulting symptoms have been classified as myofascial pain-dysfunction(MPD).

MPD is a stress related disorder resulting in increased masticatory muscle tension. It is associated with parafunction of the masticatory muscles (in particular, clenching) resulting in muscle fatigue and pain.

Laskin and Block suggested MPD represents a psychophysiological disease that primarily involves the muscles of mastication. They thought MPD starts as a functional disorder, progressing to organic changes within the temporomandibular joint capsules and masticatory muscles, ultimately leading to changes within the dentition. The fatigue was predominately related to psychologically motivated, persistent involuntary tension-relieving oral habits (10).

2.2 Mandibular Clenching and Masticatory Muscles

Jensen et al (12) studied toothclenching in relation to common migraine because of frequent reports of pericranial masticatory muscle tenderness during migraine attack. Their suspicion of increased muscle tension was based upon work of Tfelt-Hansen et al (13).

Tfelt-Hansen found, in patients during migraine attacks, a common symptom of pain located in the anterior temporalis region bilaterally. Injection of these

muscle areas with saline or lidocaine often resulted in immediate relief from the migraine pain symptoms.

Jensen et al (12) examined the temporalis muscle and based upon their results found no significant differences detected in muscle tenderness before of after experimental procedures. They reported, from their observations, muscle tenderness within the temporalis muscles that occurred during migraine headache was not likely caused by muscle ischemia due to tonic involuntary muscle tension. The lateral pterygoid muscles were not included in these studies. In their conclusions, a possible explanation for the pain noted within the muscles was an unphysiological strain leading to higher metabolic rates, higher intramuscular pressure, possibly edema formation and sterile inflammation resulting in the release of algogenic substances into the muscular tissue.

Earlier work by Christensen (14) suggested muscle tenderness after non-physiologic work typically required a few hours to develop. Christensen's work, at that time, represented the main experimental evidence supporting the clinical notion of MPD caused by muscle hyperactivity. The problem exhibited by these studies was the experimentally induced bruxing and resultant muscle fatigue did not necessarily hyperactivate the

muscles most frequently implicated in the syndrome; the lateral pterygoid muscles. The lateral pterygoid muscles control mandibular functions most often reported as limited in patients suffering from MPD complaints, including mandibular protrusion, lateral excursive movements, and vertical opening.

Christensen (15) in later work reported in man, maximum voluntary isometric contractions of the mandibular elevator muscles give rise to the subjective sensations of fatigue, pain, and total exhaustion of the contracting muscles. The lateral pterygoid muscles were not specifically identified but the descriptors of pain and its location strongly suggested lateral pterygoid involvement. The lateral pterygoid muscle did not receive much attention because of the difficulty in working with them due to their anatomical location. Electromyographers could not utilize surface electrodes to study this muscle group and found that insertion of needle electrodes difficult.

Scott and Lundeen (16) realized the involvement of the lateral pterygoid muscles in MPD, and created an experimental model to test it. A group of volunteers were asked to hyperactivate their lateral pterygoid muscles by vigorously thrusting their mandibles protrusively and retrusively over a period of time vs a

control group. The resultant pain pattern was consistent with that reported from patients suffering MPD, and they concluded hyperactivity of the lateral pterygoid muscles was partially responsible.

2.3 Electromyography of the Lateral Pterygoid Muscles

Lehr and Owens (17) studied the lateral pterygoid muscles electromyographically and noted the muscle were most active during protrusive movements of the mandible, including incisor clenching (the most common parafunctional clenching pattern).

The muscles were not active during molar clenching or retrusion of the mandible. They suggested the role of the lateral pterygoid muscles was for protrusive mandibular movements and that they did not function during molar clenching. They did not distinguish between molar and anterior clenching patterns but their work suggests the lateral pterygoid muscle was active during the most frequent parafunctional clenching activity.

Sessle and Gurza(18) examined the lateral pterygoid muscles and their involvement during mandibular movement. They found that both heads of the lateral pterygoid muscles showed reflex induced excitation and

silent periods as a result of stimulation applied to oral facial sites including the teeth.

These effects were thought related to mechanisms for protection and stabilization of the mandibular joint and masticatory muscles. The suggestion of the lateral pterygoid muscle group being not just an opening muscle group, but rather a muscle group active during clenching of the mandible as part of a compensatory stabilization process was important to development of the concept of the lateral pterygoid muscle group being active during jaw clenching parafunctional activity.

They suggested the action of the muscles was primarily isotonic/isometric during these parafunctional activities. The result was hypertrophy of the muscle group. The majority of the parafunctional activity was reported during the sleep hours. A suggestion for this was the proprioceptive protective reflexes within the periodontal ligaments surrounding the teeth were in effect turned off during the sleep cycle.

Juniper's work (19) further supported the notion of the lower belly of the lateral pterygoid muscle being active when depressing, protruding or deviating the mandible, and, it became active during closing and clenching in patients suffering pain.

Wood et al (3) studied the lower belly of the lateral pterygoid muscle electromyographically and reported the muscle was most active during anteriorly or contralaterally directed intercuspal clenching, vertically directed clenching, with the mandible positioned to the contralateral side or protrusively, and during jaw opening. The muscle was least active during vertically, ipsilaterally, or posteriorly directed intercuspal clenching.

Widmalm et al (20) performed anatomical and electromyographic studies and found strong to very strong activity consistently observed within the upper head of the lateral pterygoid muscles during tooth clenching and tooth gnashing. The inferior heads were noted to exhibit negligible to slight activity during ipsilateral movements or clenching but were co-activated bilaterally with strong to very strong activity during jaw opening, protrusion, swallowing, tooth gnashing and during passive retrusion. Their observations supported the notion of parafunctional activity of the lateral pterygoid musculature during tooth clenching and gnashing.

They also found that during this parafunctional activity, the lateral pterygoid muscle was most active when intercuspal clenching was directed anteriorly and

contralaterally and during eccentric clenches in a protrusive or contralateral position (3, 80).

Ligamental laxity within the temporomandibular capsules, often observed in patients suffering from atypical facial and head pain, causes the lateral pterygoid muscles to come into tone, to stabilize the condylar position, during tooth contact and during clenching.

During parafunctional jaw clenching, both the upper and lower bellies of the lateral pterygoid muscle are contracting to position the temporomandibular condyle within the glenoid fossa while the major powerful closing muscles (masseter, temporalis, and medial pterygoid) are exerting compressive forces upon the condyle against the articular disc and the glenoid fossa.

The parafunctional isotonic activity of the lateral pterygoid muscles results in hypertrophy. Hypertrophy in itself is not a significant problem but hypertrophy in combination with sustained muscle contraction results in decreased space within the infratemporal fossa. Vital structures within this space are potentially compromised.

2.4 Maxillary Artery and the Lateral Pterygoid Muscles

The maxillary artery passes through the infratemporal fossa and usually passes between the upper and lower bellies of the lateral pterygoid muscles. The effect of the contracted hypertrophied muscles upon the maxillary artery is the development of external mechanical compression creating stenosis within the lumen of the artery. This stenosis can result in ischemia due to flow disturbances.

In addition to imposed ischemia within the muscles of mastication supplied by the maxillary artery, there is potential for referred pain to the forehead and anterior scalp as a result of pressure on nerve fibers within the adventitia of the maxillary artery. (7)

Transient external compression of the maxillary artery can create vascular flow disturbances with development of turbulence distal to and perhaps proximal to the site of compression. The flow disturbance within the maxillary artery could affect not only the muscles of mastication but also generate ischemia within the cranium via diminished flow into the middle meningeal and accessory meningeal arteries affecting the dura and the gasserian ganglion.

2.5 Pain Genesis From the Maxillary Artery

Innervation of dural arteries is composed of both vasomotor efferents (autonomic) and sensory afferent fibers. (8,9) Mechanical stimulation of these dural arteries can result in pain. Diminished flow into the accessory meningeal artery is important as ischemia within the gasserian ganglion may result in modified neurological function of the trigeminal nerve.

Moskowitz et al (34) suggested cephalic blood vessels (pia and dural) were the most important source of all headaches and are innervated by sensory fibers that arise from ganglia (gasserian) innervating the forehead, scalp and neck. These sensory fibers contain vasoactive peptides which are released from peripheral (perivascular) and central terminations to mediate activation of a final common pathway that they termed trigeminovascular system.

Moskewitz later reported that chemical mediators may develop within the brain parenchyma, the blood vessel walls and perhaps the blood itself (25). He suggested unknown triggers stimulate the trigeminovascular axons resulting in pain and effecting release of vasoactive neuropeptides from perivascular axons. These released neuropeptides activate endothelial cells, mast cells,

and platelets to increase the extracellular levels of amines, arachidonate metabolites, peptides and ions. Hyperalgia and prolongation of pain develop as a consequence.

Dostrovsky et al found stimulation of the middle meningeal artery and superior sagittal sinus of cats produced a response in neurons within the trigeminal nucleus (33). They also found almost all of these same neurons receive convergent input from the facial skin and the periorbital region. The neurons were classified as nociceptive and characteristics of their cerebrovascular activation was consistent with mediating vascular head pains, with typical referral zones to the periorbital and anterior temporal regions in man. The report also described preliminary results of recordings from rat trigeminal ganglion neurons activated by electrical stimulation of the middle meningeal artery and sagittal sinus.

2.6 Pain Genesis and the Trigeminal Nerve

De Marinis et al reported on reduction of head pain and facial vasodilatation in patients who had gasserian ganglion lesions performed. They found with patients who suffered from headache, that 50% were pain free on the side of the trigeminal lesion. The reduction in

pain was not, in association, hypoesthesia resulting from the lesion. There was a decrease in vascular responses (histamine induced facial flushing and increase in temperature) on the operated side. They concluded the trigemino-vascular system was involved in control of headache of a vascular type and associated craniofacial vasodilatation in human subjects.

Trigeminal nerve pathways to the cerebral arteries were found to have intracavernous branches traced from the ophthalmic nerve (V_I) (4, 32). These branches joined the cavernous plexus and distributed forward with autonomic nerves to the adventitia of the internal carotid artery emerging from the sinus with the artery. A strong recurrent branch from the plexus joined the Abducent Nerve (VI) and passed posteriorly leaving the nerve at the pontine level to innervate the basilar artery and caudal circle of Willis.

In the pterygopalatine fossa, the orbitocilliary branch of the maxillary division of the trigeminal nerve (V_{II})gave off filaments that re-entered the cranial cavity through the medial infraorbital fissure and joined the cavernous plexus. The implication is irritation of the gasserian ganglion may give rise to altered flow to the cerebral cortex.

A population of neurons in the brainstem trigeminal complex was discovered (29) that could be activated by stimulation of major dural blood vessels. The dura-responsive neurons exhibited response properties that were appropriate for a role in the mediation of vascular head pain. The neurons were found to exhibit nociceptive facial receptive fields whose periorbital distribution was similar to the region of referred pain evoked by dural stimulation in humans. Dura-responsive brainstem trigeminal neurons may have a role in the mediation of dural vascular head pain and also indicate that such neurons may contribute to nociceptive processing within the dorsal horn (29).

Visocchi et al studied cerebral blood flow velocities and trigeminal ganglion stimulation and found during unilateral trigeminal ganglion stimulation the flow velocity within the cerebral arteries significantly decreased unilaterally (31).

The suggestion of the lateral pterygoid muscle exhibiting tonic activity during parafunctional jaw clenching and bruxism allows development of a hypothesis of its involvement in generation of ischemia within the infratemporal fossa and intracranially affecting both the dura as well as the posterior

cerebral circulation. Due to the unique anatomy of the infratemporal fossa including the passage of the maxillary artery and its relationship of the lateral pterygoid muscle heads, it is speculated the maxillary artery may be subjected to compressive forces during parafunctional mandibular clenching.

CHAPTER 3.

MATERIALS AND METHODS

3.1 Population Sample

Two populations of patients were included in this investigation. The examination form utilized is presented in Appendix II. The form represents 74 measurements and the data is expressed as a ratio of X/74.

The first group represented a control group displaying no overt symptoms of craniocervical myofascial pain detectable by examination. This group all demonstrated a score of 0 or 0/74. Five asymptomatic volunteers (4 male and 1 female, mean age 19 years; range 15 - 20 years) were examined clinically.

The second population included those patients who demonstrated signs of muscle palpation pain, limitation of mandibular movements, headache, facial pain and fatigue of the masticatory musculature. This group (N=46, mean age 30 years; range 16 to 57 years, N=36 females and N=10 males) were selected on the basis of positive craniomandibular pain indices as per the examination form.

Both populations were selected as new patients who presented to my practice over a period of 4 months. The protocol for examination and inclusion in the study

has been given approval by the institutional ethics committee and all subjects gave written consent to the procedures (Appendix IV).

All volunteers were examined with digital palpation of the masticatory muscles, range of mandibular movements including border movements, and signs of intracapsular temporomandibular noise. Normal volunteers were rejected if they exhibited signs of masticatory muscle tenderness to palpation. Symptomatic volunteers were selected who demonstrated positive palpation symptoms in addition to suffering from headache and facial pain.

3.2 Magnetic Resonance Imaging

The selected volunteers were all imaged utilizing magnetic resonance imaging (MRI) initially to examine the anatomy of their infratemporal fossae bilaterally. The purpose of the MRI examination was to ensure to location of the maxillary artery within the window of the coronoid notch so that it could be examined with ultrasound. The relationship of the maxillary artery and the lateral pterygoid muscles was assessed to anatomically located the artery between the upper and lower pterygoid bellies.

Prior to the MRI, the chosen volunteers were given a questionnaire regarding previous surgeries and potential of any foreign metal placement or accidental foreign metal implantation into their bodies to determine contraindications to MRI procedures.

3.3 MRI Technique:

The volunteers were placed supine on the scanner gantry bed and 8.5 cm bilateral surface coils were positioned over the area of the infratemporal fossae with the center of the coil over the area of the coronoid notch of the mandible.

All volunteers were imaged with a 1.5T Philips MRI utilizing T1 weighted sequence (TR=250 TE=30, 1 measurement 100% acquisition, 4 slices 1 cm thick) axial scout images. The axial scout images were made to plan the slice orientation to cephalometrically correct the slices so that they were perpendicular to the medial lateral poles of the temporomandibular condyles.

The planned images were made in the sagittal and coronal planes (FOV=100 mm, 256X256 matrix, 4 measurements at 80% acquisition; TR=100, TE=30,80; 6 slices 3mm thick, 1.2mm slice factor). The position of the TMJ discs were ascertained to ensure no closed lock internal

derangement and the location of the maxillary artery was noted particularly in its relationship with the upper and lower bellies of the lateral pterygoid muscles.

The images obtained were examined and the cranial caudal dimension of the upper and lower bellies of the lateral pterygoid muscles were measured at mid-belly. These numbers were tabulated and then ratios of the lower belly dimension to upper belly dimension were calculated. The error of the measurement was less than 10% (as reported by Philip's in their technical manual) and was considered negligible following ratio calculation.

3.4 Ultrasound Imaging

All volunteers were examined using diagnostic ultrasound imaging through the coronoid notch window to locate the maxillary artery. Color flow doppler sonograms as well as spectral doppler imaging was performed on both right and left maxillary artery with the patient resting with their teeth separated and then with their teeth tightly clenched together.

A 10 cm diameter 1.5 cm thick gel pad was placed over the area of interest to improve contact of the transducer with the skin surface to improve resolution

of the artery. A linear array 5 megahertz rectilinear transducer (1.5 cm X 8 cm.) was positioned over the coronoid notch. The maxillary artery was identified and the doppler angle was corrected to the direction of flow within the maxillary artery.

The imaging was performed with the volunteers lying on their sides. This posture was the most common noted sleeping posture stated by each volunteer. This lying posture generates pressure on the mandible that allowed lateral excursion of the temporomandibular joints and placed the volunteer's contralateral temporomandibular joint into a protrusive posture bringing both lower bellies of the lateral pterygoid muscles into tone. The contralateral artery was imaged.

The volunteers were given a surgical latex rubber glove and were instructed to place three fingers of the glove together and then to place the layered fingers of the glove along the ipsilateral occlusal plane of the anterior and posterior teeth of the maxillary artery being examined. They were then instructed to lay with their teeth just apart separated by the latex rubber. They were told to allow their jaw to relax and not to bring pressure to their teeth.

The use of the interocclusal rubber was necessitated because all volunteers found it extremely difficult, and in some cases impossible, to sustain maximum clenching of their teeth for a period of 60 seconds due to development of pain within the teeth and activation of the periodontal proprioceptive protective reflex.

The interpositional material interrupted the reflex for the appointed examination time.

Typically, the volunteers during sleep are able to sustain clench times in excess of the experimental time used for this study due to cessation of the protective proprioceptive periodontal reflex during sleep.

The maxillary artery was identified by its relative size in comparison to the other arterial flow within the coronoid notch window and by its anatomical position anterior to the condyle. The superficial temporal artery was identified to ensure that it was not mistakenly assessed. All images were videotaped for later evaluation. The images were also printed on film.

3.5 Doppler Imaging

Color flow doppler imaging was utilized as it allowed depiction of blood flow through the entire vessel lumen,

rather than in a single isolated location. The flow characteristics throughout the lumen were continuously displayed and localized abnormalities were readily apparent. These abnormalities provided information on the location of flow abnormality and allowed accurate placement of the doppler sample volume at the location of the abnormality for quantitative flow assessment.

The limitations of color flow doppler alone include:

1) flow information is qualitative and not quantitative. The information is based on average doppler shift rather than peak shift.

2) the doppler information is not corrected for the doppler angle. The doppler angle is the angle between the doppler ultrasound beam and the flow vector or the vessel lumen. The doppler angle is necessary to convert doppler shift to flow velocity.

3) The shades of color are very coarse representations of velocity differences within the flow stream. (22)

Following location of flow abnormalities as seen with color flow doppler, doppler imaging was performed of the maxillary artery during rest, then during clenching and then again during rest. The lumen of the maxillary artery was measured utilizing the measurement grid available on the machine. Three measurements were made of each. It is noted that the error related to ultrasound imaging in vessels this small is $\pm$ 2 mm.

Following measurements of the changes in vessel lumen diameter, the doppler angle was adjusted and doppler waveforms of arterial flow within the maxillary artery were made with the maximum velocity recorded when the volunteer was resting with their teeth apart.

The volunteer was then instructed to clench their teeth tightly together for a period 60 seconds and the waveform velocities were recorded noting specifically the development of jet waveforms and then development of disturbed and turbulent flow. Following the 60 second clench time a maximum velocity waveform was recorded.

The volunteer was then asked to relax their clenching posture of their jaws and to separate their teeth slightly as they did during recording of the flow velocities initially. The transducer was kept in place and the flow velocities were recorded for a period of 5

minutes following relaxation of the masticatory muscles monitoring the flow.

The patients were allowed 15 minutes between examinations to allow flow to normalize within the maxillary artery prior to examination of the contralateral artery. This time seemed to be optimum for all volunteers as longer periods of time did not alter the rest velocity of the contralateral side.

The spectral doppler imaging was recorded and the images were examined to test for spectral changes and the data that was obtained was tabulated and graphed to examine for any trends regarding age, craniomandibular pain indices, ratio of lateral pterygoid mass, lumen diameter change at rest and during clenching, and peak flow velocities during rest as well as during clenching.

CHAPTER 4.

RESULTS

AND

DISCUSSION

The question this study attempted to answer was: did the lateral pterygoid muscle belly hypertrophy and contraction adversely affect the lumen diameter of the maxillary artery and consequently alter blood flow dynamics? The net result of this altered blood flow would be generation of pain due to possibly ischemia particularly within the distal segment of the artery (past the area of stenosis) and its tributaries.

4.1 Population

The age distribution of the symptomatic group ranged from 16 years to 57 years of age. The mean was 36 years with a standard deviation of 1.5. The majority of the volunteers were in the 25 to 45 year group. Reports in the literature suggest that myofascial pain seems to effect patients primarily within the second to fourth decade of life (10,16,100,101). The age distribution of the normal group ranged from 15 years to 20 years of age, the mean was 18 years with a standard deviation of 1.8.

The literature further reports that persons who suffer from these types of pain and dysfunction appear to have a strong predilection for females over males. The literature reports ratios as high as 10:1 female to male. The distribution of symptomatic volunteers in

this particular study had ratios of female to male of approximately 4:1. The normal volunteers in spite of their low numbers demonstrated a female to male ratio of 4:1. This reverse trend is suggestive that there are more normal males than females.

The population of symptomatic volunteers were all selected based upon the criteria of pain within the head and facial structures. The selection process was based upon clinical examination and generation of a craniomandibular pain index (Appendix II). The volunteers were selected from my private practice who were referred for treatment of prominent pain and dysfunction of their craniomandibular apparatus.

The CMI Pain Index was scored by the number of positive examination results out of a possible 74 and then a ratio was calculated. This ratio was tabulated and compared to age distribution. Due to the unique nature of my private practice from which these volunteers were selected, they were not age matched. All volunteers suffered from marked pain and dysfunction that was both acute and chronic in nature. The symptomatic volunteers demonstrated a trend of relatively high CMI scores.

Table 1.

CMI Pain Indices

Patient Number	Age	CMI Score	Ratio/1
1	54	53/74	0.720
2	24	62/74	0.840
3	35	68/74	0.920
4	34	66/74	0.890
5	57	68/74	0.920
6	40	59/74	0.800
7	23	65/74	0.880
8	28	58/74	0.780
9	24	67/74	0.910
10	31	55/74	0.740
11	26	63/74	0.850
12	39	65/74	0.880
13	46	66/74	0.890
14	27	64/74	0.860
15	35	62/74	0.840
16	21	67/74	0.910
17	53	51/74	0.690
18	32	62/74	0.840
19	27	67/74	0.910
20	44	60/74	0.810
21	33	63/74	0.850
22	31	68/74	0.920
23	38	63/74	0.850
24	28	60/74	0.810
25	46	65/74	0.880
26	16	61/74	0.820
27	38	56/74	0.760
28	31	64/74	0.860
29	28	63/74	0.850
30	46	60/74	0.810
31	52	65/74	0.880
32	32	61/74	0.820
33	42	58/74	0.780
34	23	65/74	0.880
35	43	65/74	0.880
36	30	67/74	0.910
37	36	63/74	0.850
38	56	60/74	0.810
39	34	54/74	0.730
40	57	61/74	0.820
41	23	67/74	0.910
42	45	50/74	0.680
43	41	55/74	0.740
44	43	69/74	0.930
45	39	64/74	0.860
46	27	67/74	0.910
Mean ± S.D.	36 ± 10.45		0.84 ± 0.065

4.2 MRI Results

Magnetic resonance images were obtained for two reasons. The first was to anatomically relate the lateral pterygoid muscles within the infratemporal fossa. The maxillary artery was identified and its unique anatomical position between the upper and lower belly of the lateral pterygoid muscles was identified.

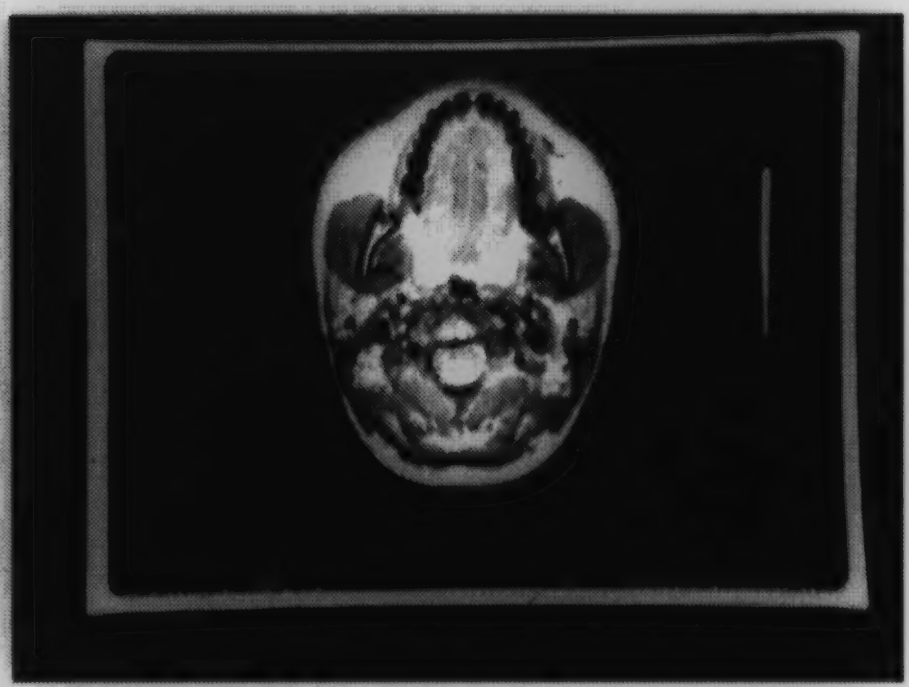


Figure 1.

Axial MRI Illustrating the Lateral Pterygoid Muscles (a)

The anatomical location of the lateral pterygoid muscles was depicted in Figure 1. This image is an axial projection at the level just inferior to the mandibular condyles. The lateral pterygoid muscles (a) can be easily visualized and their mass can be appraised but accurate measurements cannot be made with these particular images. Higher resolution surface coil images would be required to make such measurements to clearly define the borders of the lateral pterygoid muscle.

MRI was used to demonstrate the location of the lateral pterygoid muscles in the coronal plane. This plane offers better appreciation of the bulk of the muscle anterior to the mandibular condyles. From the coronal image, (Figure 2), one cannot isolate the upper and lower bellies of the lateral pterygoid muscle group. The use of high resolution surface coils would be required to resolve the issue of muscle size.

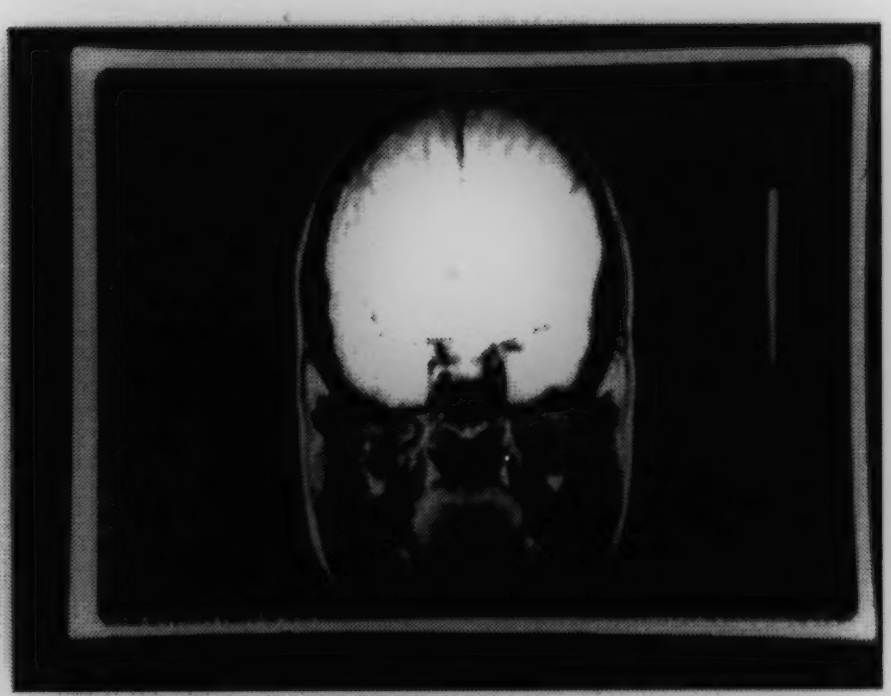


Figure 2.

Coronal MRI. The slice is just anterior to the temporomandibular condyles.

a: Lateral Pterygoid Muscle upper belly

b: Medial Pterygoid Muscle

The sagittal MRI is the best image for viewing the lateral pterygoid muscle and the maxillary artery. The image seen in Figure 3. clearly demonstrates the bulk of the lateral pterygoid mass as well as the maxillary artery.

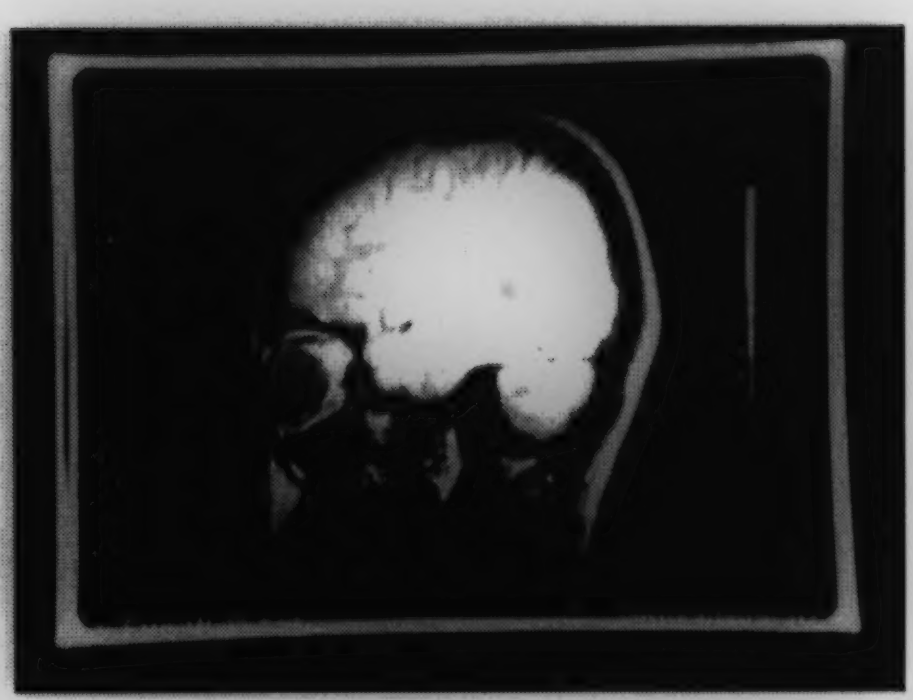


Figure 3.

Sagittal MRI taken just medial to the temporomandibular condyles. The attachment of the lateral pterygoid to the mandible at the pterygoid fovea is not demonstrated with this particular image.

a: Lateral Pterygoid Muscle lower belly

b: Maxillary Artery and Vein

The MRI image most useful in this study was the view of the temporomandibular condyle and the infratemporal fossa. This particular image allowed measurement of the upper and lower belly of the lateral pterygoid muscle

and demonstrated the location of the maxillary artery as it passed between the two bellies of the muscle.

Figure 4.

Sagittal MRI of the TMJ, Upper and Lower Bellies of the Lateral Pterygoid Muscles, and the Maxillary Artery.

This particular image is a typical view noted of all the volunteers. Both the upper (b) and lower bellies (a) of the lateral pterygoid muscle are clearly demonstrated and the maxillary artery (c) can be visualized arising from the external carotid artery. The maxillary artery was observed extending medial to the mandibular condylar neck and lower belly of the lateral pterygoid muscle.

The maxillary artery was additionally observed at the antero-superior margin of the lower belly of the lateral pterygoid muscle indicating its path between the two bellies of the lateral pterygoid muscle.

It was from these MRI images that I measured the muscle mass of the upper and lower bellies of the lateral pterygoid muscles. The measurement data was placed into two tables. The first table represents the measured muscle mass of the normal population included in this study.

Table 2.

Lateral Pterygoid Belly Measurements: Normal

Subject	Lt upper lat. ptery.mm	Lt lower lat. ptery.mm	Rt upper lat. ptery.mm	Rt lower lat. ptery.mm	Ratio Lt lower/ upper	Ratio Rt lower/ upper
1	6	9	5.5	9	1.5	1.64
2	4.5	7.5	5.0	8.0	1.67	1.60
3	6.0	10.0	6.0	9.0	1.67	1.5
4	7.0	12	7.0	11.0	1.71	1.57
5	7.0	12	6.0	11.0	1.71	1.83
Mean	6.1	10.1	5.9	9.6	1.65	1.62

Due to the difficulty in finding normal volunteers based upon my selection criteria, I found that the majority of volunteers were very young. The muscle mass ratios measured were all less than 2:1. The suitability of the normal population was inadequate for studying the relationship of pain and muscle hypertrophy as the population was all very young and there were

insignificant numbers. The ratios observed in the normal population of less than 2:1 was within the range reported within the literature (1,3,7,17,20).

The difficulty of obtaining a wider range of age in the normal population was partly due to the selection criteria. I decided the normal population should demonstrate no signs of craniomandibular dysfunction and experience no pain related to these structures. This limitation excluded a great many persons from this study.

The symptomatic volunteer MRI's were measured and there was noted ratios that were all above 1.7 for the two lateral pterygoid bellies and these ratios could not be related to physical stature of the volunteer.

The density of the lateral pterygoid muscles noted in the MRI images, demonstrated no signs of fatty infiltration suggestive of atrophy but rather indicated a toned muscle.

Table 3.

Lateral Pterygoid Belly Measurements: Symptomatic

Subject #	Lt Upper Lat. Ptery.mm	Lt Lower Lat. Ptery.mm	Rt Upper Lat. Ptery.mm	Rt Lower Lat. Ptery.mm	Ratio Lt lower/ upper	Ratio Rt lower/ upper
1	7	18	9	22	2.57	2.44
2	9	22	10	21	2.44	2.10
3	8	21	8	19	2.44	2.38
4	10	23	10	21	2.30	2.50
5	8	19	9	21	2.38	2.33
6	7	16	7	18	2.29	2.57
7	8	22	7	24	2.75	3.43
8	6	16	6	15	2.67	2.50
9	7	17	8	20	2.43	2.50
10	8	15	8	19	1.88	2.38
11	6	14	8	17	2.33	2.13
12	7	16	7	17	2.29	2.43
13	7	18	8	20	2.57	2.50
14	6	15	8	18	2.50	3.00
15	8	15	9	16	1.88	1.78
16	6	16	7	20	2.67	2.86
17	7	17	6	15	2.43	2.50
18	7	18	8	18	2.57	2.25
19	4	13	5	15	3.25	3.00
20	9	20	8	17	2.22	2.13
21	8	19	9	19	2.38	2.11
22	7	16	7	17	2.29	2.38
23	5	14	6	19	2.80	3.17
24	6	15	7	15	2.50	2.14
25	8	16	8	16	2.00	2.00
26	6	13	7	12	2.17	1.71
27	8	20	8	20	2.50	2.50
28	6	14	7	14	2.33	2.00
29	7	14	7	14	2.00	2.00
30	5	13	6	13	2.60	2.17
31	7	16	7	15	2.29	2.14
32	6	14	7	16	2.33	2.29
33	8	20	8	20	2.50	2.50
34	7	16	6	16	2.29	2.67
35	6	14	6	14	2.33	2.33
36	5	14	6	15	2.80	2.50
37	8	19	7	20	2.50	3.00
38	7	15	7	15	2.14	2.14
39	6	15	6	18	2.50	3.00
40	7	15	5	13	2.29	2.60
41	6	15	7	15	2.50	2.14
42	5	15	6	15	3.00	2.50
43	8	20	8	19	2.50	2.38
44	7	19	7	21	2.71	3.00
45	8	18	6	15	2.25	2.50
46	7	20	8	20	2.86	2.50
Mean ±SD	N/A	N/A	N/A	N/A	2.44±.27	2.44±.36

The main hypothesis of the study was to demonstrate hypertrophy of the lateral pterygoid muscle mass and to test its effect upon the maxillary artery in the area of the infratemporal fossa. MRI was not suitable for examination of the size of the maxillary artery. The MRI is not a dynamic imaging vehicle and thus the artery size could not be assessed during active clenching.

4.3 Ultrasound Imaging

Ultrasound and color flow ultrasound provided the necessary imaging to assess the artery diameter and the effects of mechanical compression during clenching of the mandible.

4.3.a Ultrasound Measurement of the Maxillary Artery

The following set of three figures are typical and demonstrate in one volunteer the change in arterial diameter during the rest state when the teeth were apart, followed by the clench state with the teeth tightly occluded and then again during the rest state when the teeth were separated. The first figure (figure 5) demonstrated the arterial lumen dimension at 3 mm. The difficulty with these measurements was the error inherent in the ultrasound device itself. Resolution of such small vessels represented a error margin of $\pm$ 2 mm.

The sonogram indicated the depth of the maxillary artery at approximately 2 centimeters below the skin surface. I performed extensive anatomical dissections to correlate the MRI images and the ultrasound images to help clarify the anatomy of the infratemporal fossa. The measurements of for all symptomatic volunteers demonstrated the arterial depth within 0.5 cm the noted 2.0 cm depth for this particular volunteer. The cadaver information demonstrated the depth of approximately 2 ± 1 cm for the maxillary artery.

Figure 5.

Arterial Lumen Diameter Measurement - Rest State

After 60 seconds of clenching the arterial dimension for this volunteer was measured and was noted to be 1.5 mm. The confirmation of the artery also changed in response to the mechanical compression from the lateral pterygoid muscles.

Figure 6.

Arterial Lumen Diameter Measurement - Typical Volunteer Clench State

Following the 60 seconds of experimental clenching and relaxation with the teeth separated, the artery was re-measured and it was observed to regain some of its

initial dimension but did not return to its pre-clench state. Three measurements were made for each volunteer during this portion of the study and there was equal consistency in all three measurement sets.

Figure 7.

Arterial Lumen Diameter Measurement - Typical Volunteer

Rest State, Teeth Separated

Following the 60 seconds of experimental mandibular clenching, this particular volunteer demonstrated a luminal diameter of only 2.2 mm and did not have her

maxillary artery diameter return to its pre-clench state for over 15 minutes. This was found in those volunteers who demonstrated the greatest pain dysfunction ratios.

4.3.b Color Flow Doppler Imaging - Typical Volunteer

The maxillary artery was also imaged in each volunteer with color flow ultrasound imaging. The following set of three figures of one of the volunteers more clearly depict the artery confirmation during rest, following 60 seconds of mandibular clenching and relaxation again.

The color flow ultrasound image (figure 8) demonstrated the right maxillary artery. In this image, the volunteer had their teeth separated with the interpositional latex between their teeth. Their jaws were relaxed with no tensing of the masticatory muscles.

The color noted represented flow toward the transducer as noted by the red color. The appearance of the yellow within the artery image represents a high velocity jet being present.

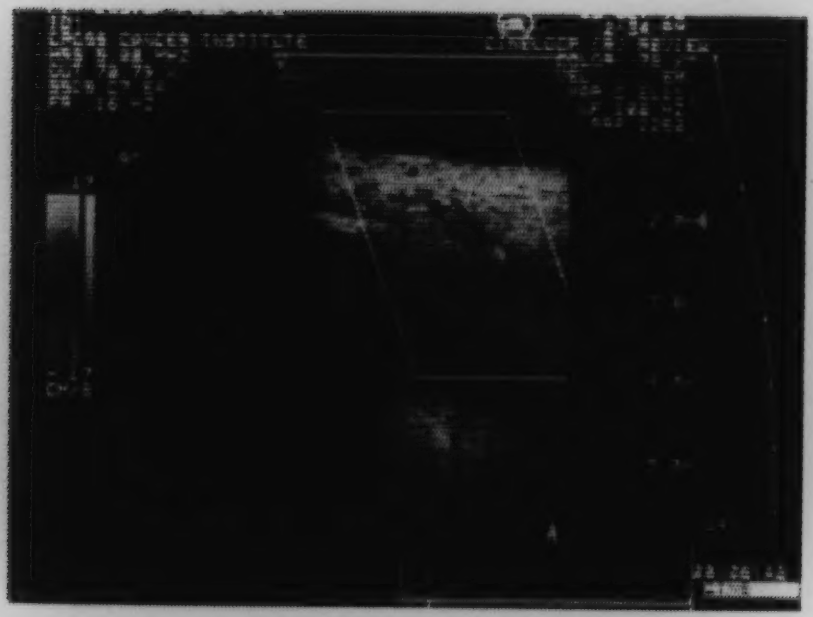


Figure 8.

Color Flow Doppler - Typical Volunteer

Rest State

When this volunteer was instructed to clench the mandibles as tightly as possible, there was a notable change in the confirmation of the maxillary artery lumen (figure 9). The luminal dimension decreased and signs of flow alaising were noted. The change noted within the lumen diameter was thought suggestive of external compression creating intra-luminal stenosis.

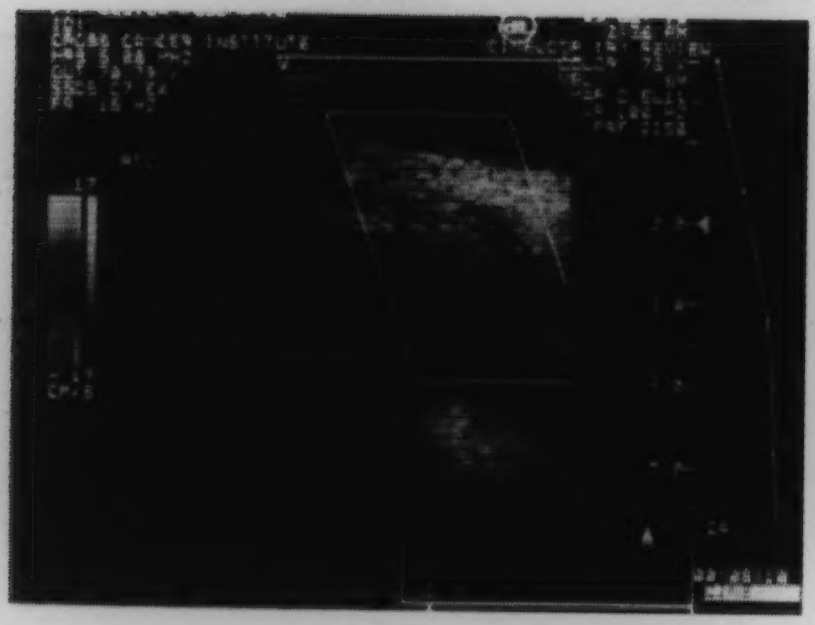


Figure 9.

Color Flow Doppler - Typical Volunteer

Clench State

The final ultrasound image for this particular volunteer demonstrated the maxillary artery following relaxation of the mandibular clenching. The vessel diameter returned to its original confirmation with disappearance of flow alaising. There is again the appearance of a high velocity jet within the lumen of the flow pattern as depicted by the yellow color.

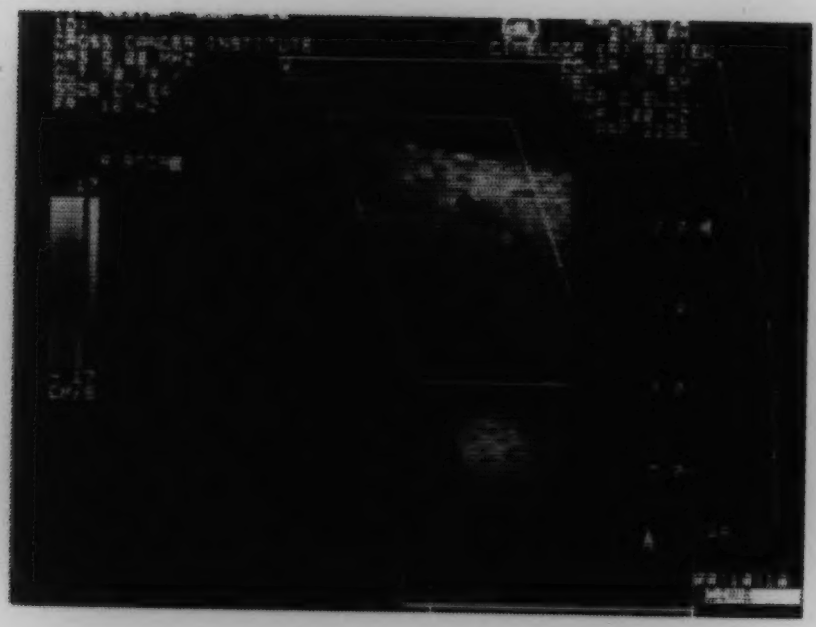


Figure 10.

Color Flow Doppler - Typical Volunteer

Relax State, Teeth Separated

With the use of the ultrasound imaging, the maxillary arteries were measured for each volunteer. The measurements included the right and left maxillary artery at rest, during clenching and then again at rest.

The normal population demonstrated no change in their arterial dimension from the initial measured rest diameter. There was no effect from clenching thus the diameter remained constant.

The reduction in luminal diameter during mandibular clenching and the muscle volume ratios measured for each volunteer seem to demonstrate a trend. It suggested muscle ratios greater than 1.7:1 in this particular group of volunteers demonstrate luminal reduction during clenching. The normal volunteer population all had muscle ratios less than 1.7:1 and no luminal changes were recorded during mandibular clenching. There were not sufficient normal volunteers to suggest if the ratio of 1.7:1 was significant.

4.4 Spectral Doppler Measurements:

The doppler waveforms were recorded and analyzed for each volunteer. The rest waveforms were measured for peak velocity. The waveform was analyzed for confirmation of the pattern and for signs of flow disturbance.

4.4.a. Spectral Waveform Change From Rest to Clench to Rest Again - Typical Volunteer

The following group of five images (figure 11-16) were typical and were included to demonstrate the disturbance of flow starting with a clear sharp waveform that during mandibular clenching demonstrates early signs of stenosis as the peak velocity measured raised above the

resting peak velocity. This increase is due to the Bernoulli effect as the lumen diameter narrows. As the clenching continued, the peak velocity measured started to drop as the disturbance of the waveform was more apparent. Disturbance included spectral broadening with loss of the spectral window.

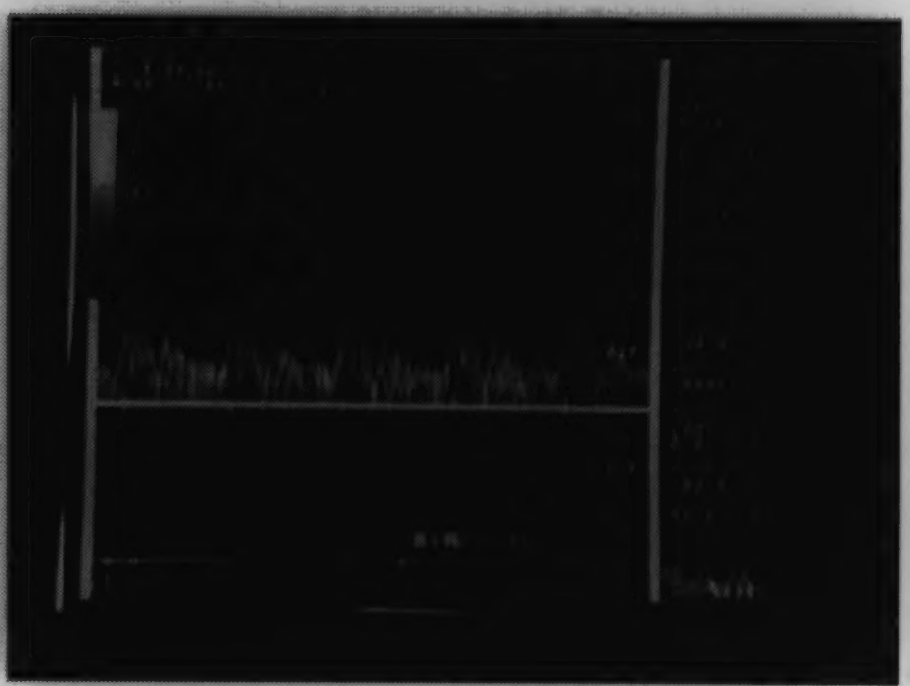


Figure 11

Spectral Waveform -Typical Volunteer

Rest State

Figure 11 demonstrated a resting waveform with a peak arterial velocity recorded at 65.4 centimeters per second. The spectral waveform demonstrated a sharp border with a well defined spectral window.

This volunteer was instructed to clench their teeth tightly together and another waveform was recorded 15 seconds into the clench cycle. The maximal velocity increased to 85.4 centimeters per second with noted spectral broadening(figure 12). The window decreased demonstrating increased numbers of different velocities present (delamination of flow) suggestive of flow disturbance and the jet velocity was suggestive of development of arterial lumen stenosis.

Thirty seconds of clenching yielded further broadening of the spectral border with loss of the window (figure 13). The velocity decreased to 77.1 centimeters per second. As the stenosis increases, the peak velocity decreases.

Figure 12.

Spectral Waveform - Clenching Started

Peak Velocity Increased

Further clenching yielded further peak velocity reduction to 48.7 centimeters per second. The spectral waveform demonstrated further disruption with signs of bruit development which was thought to represent vibration of the arterial walls (figure 14). The appearance of the waveform lacks definition and is ragged.

Following the 60 second examination time, the final peak velocity recorded was 32.5 centimeters per second. The spectral diagram demonstrated disruption of the spectral border with no jets noted (figure 16). The appearance of the border is ragged and there is total interruption of flow suggesting intermittent occlusion.

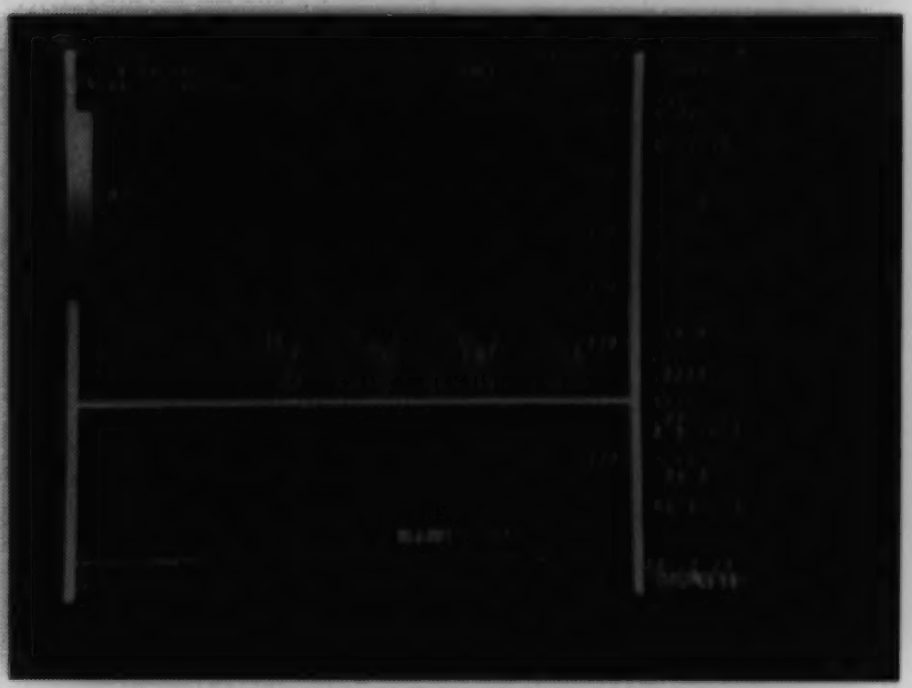


Figure 13.

Spectral Waveform - 30 Seconds of Clenching

Peak Velocity Starting to Diminish

Following 30 seconds of mandibular clenching, the peak velocity diminished to 77.1 cm/sec. The spectral window has disappeared and the border is blurred.

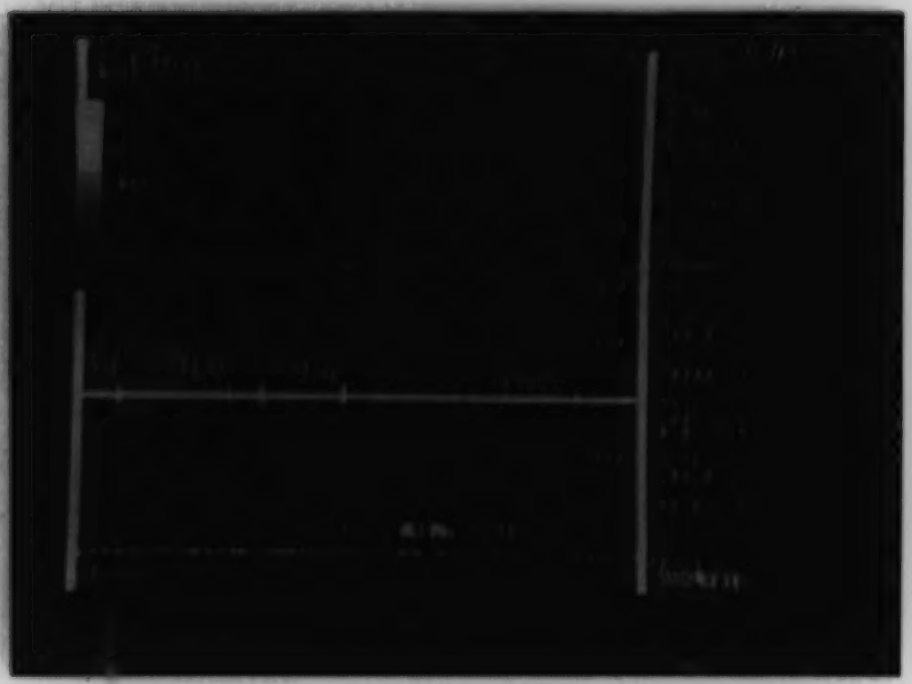


Figure 14.

Spectral Waveform - 45 Seconds of Clenching

Flow Disturbance

Following 45 seconds of mandibular clenching, the peak velocity had diminished to 48.7 cm/sec. The stenosis was thought to be increasing due to the diminishing peak velocity.

Figure 15.

Spectral Waveform - 60 Seconds of Clenching

Loss of Spectral Border, Flow Disturbance

Following 60 seconds of mandibular clenching, the spectral waveform exhibits signs of significant disruption of flow. The peak velocity has diminished to 32.5 cm/sec. The spectral border is indistinct.

4.4.b Severe Stenosis

The next group of images of a volunteer were selected to demonstrate a more severe stenosis with partial

occlusion of the maxillary lumen. This volunteer, in spite of being able to generate intermittent occlusion within the maxillary artery, still following relaxation was able to re-establish a normal doppler waveform.

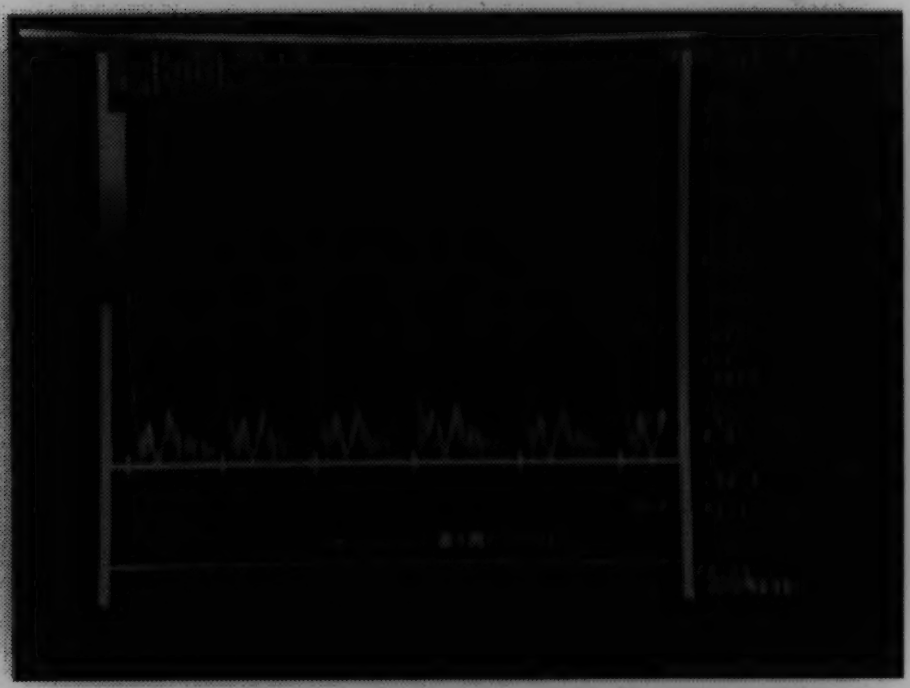


Figure 16.

Spectral Waveform - Sharp Border

Window Present

This was a spectral waveform at rest with a peak velocity measured at 88.6 cm/sec. There was a sharp spectral border with a well defined spectral window.

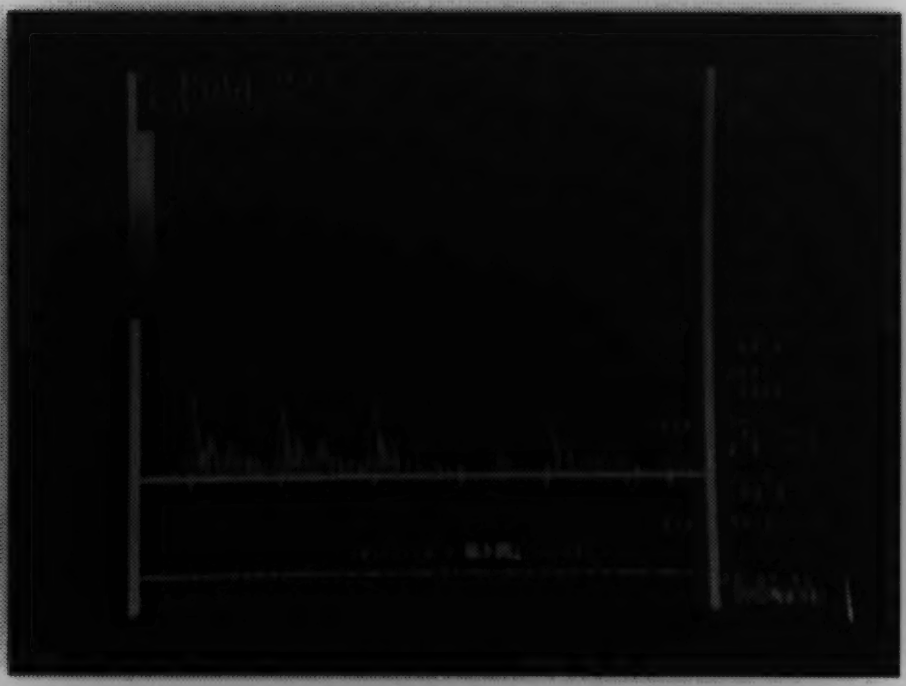


Figure 17.

Spectral Waveform - Spectral Broadening

Window Diminished

This image was made following 45 seconds of mandibular clenching. Signs of retrograde flow were visible and these was signs of spectral broadening suggestive of flow disturbance. The spectral window has all but disappeared.

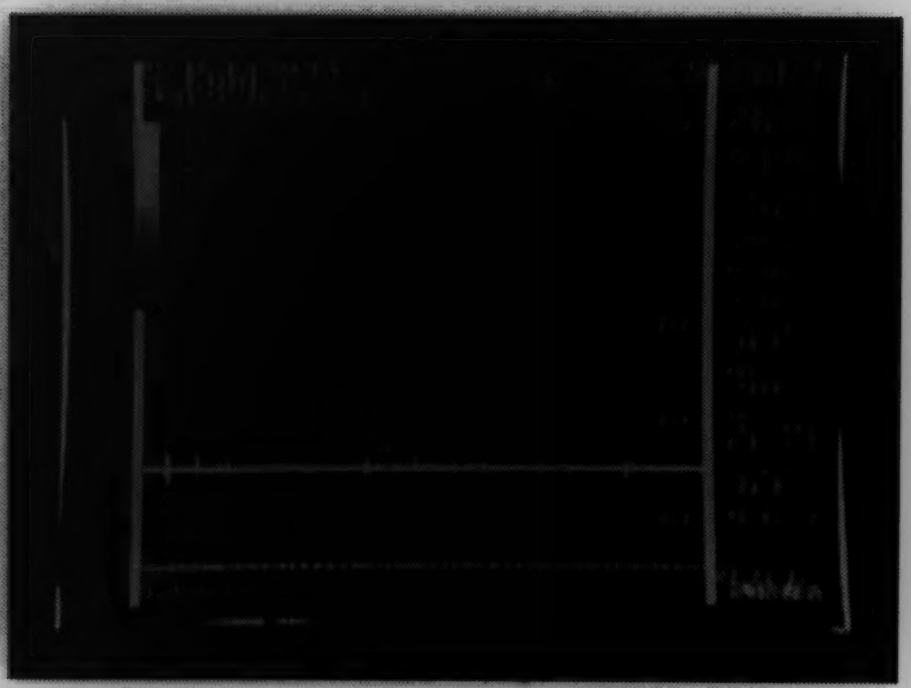


Figure 18.

Spectral Waveform - Signs of Occlusion with Presence of Bruits

Following 60 seconds of experimental clenching, the spectral trace demonstrated signs of arterial occlusion as evidence by intermittent pulses of flow and bruit formation. At this point in time during this examination, the volunteer had to stop and relax due to significant pain production.

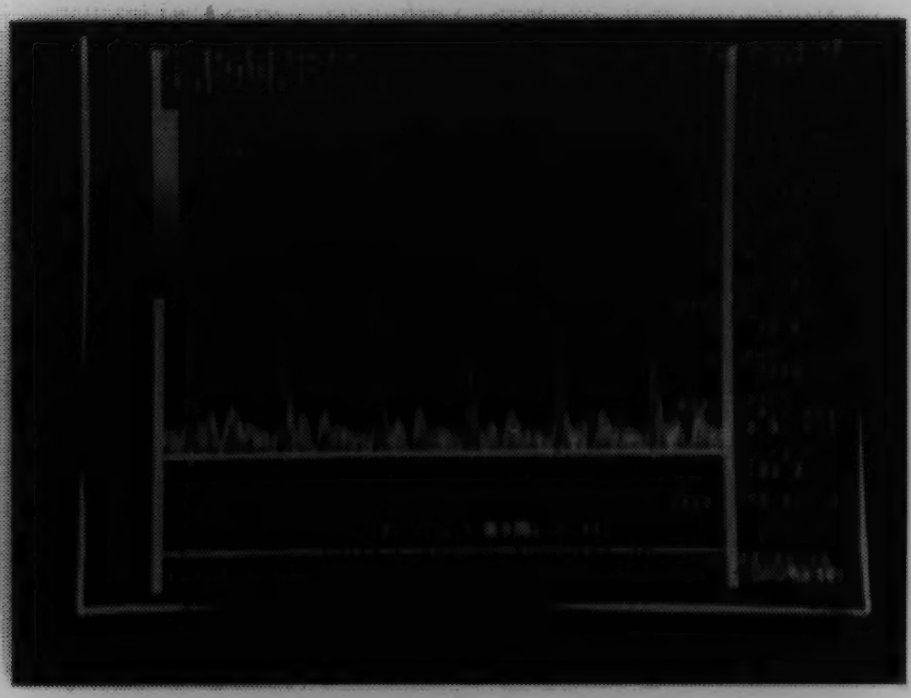


Figure 19.

Spectral Waveform - Relaxed State

Return of Normal Spectral Pattern

Following relaxation of the mandible, the peak velocity measured increased to 63.9 cm/sec. The spectral border and width returned to pre-clench conditions. There was re-appearance of the spectral window.

4.4.c. Significant Peak Velocity Reduction and Spectral Disturbance

The following two images of another volunteer were included to demonstrate dramatic reduction of peak flow velocity and disruption of the spectral border.

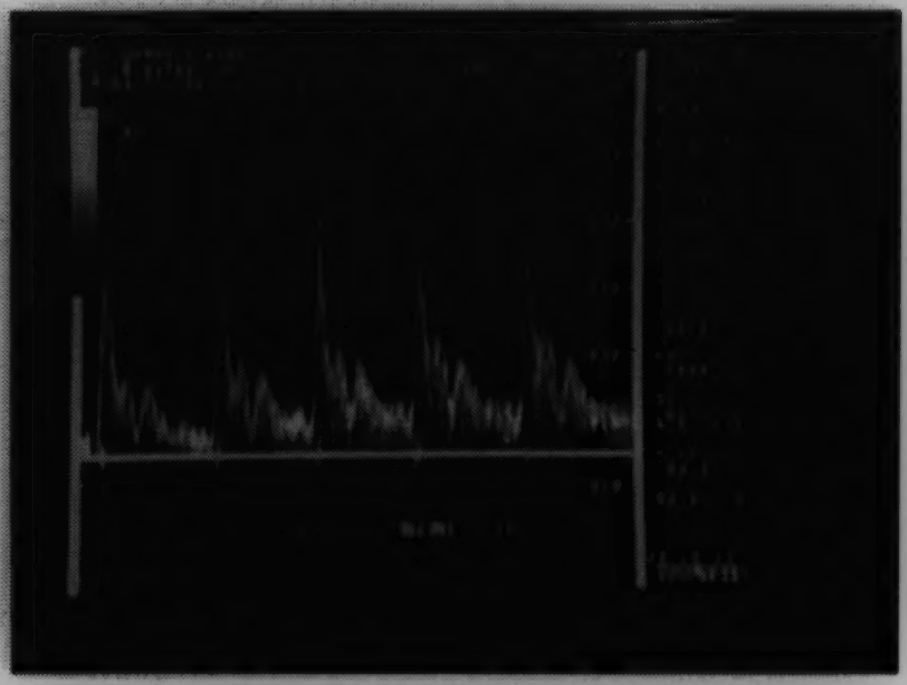


Figure 20.
Spectral Waveform - Rest State
Sharp Border, Window Present

The spectral border is sharp with a well defined window. The peak velocity was measured at 70.5 cm/sec.

Figure 21.

Spectral Waveform - Clenched State

Loss of Border Definition, Peak Velocity Markedly Reduced

Following 60 seconds of experimental mandibular clenching, the spectral border is indistinct with spectral broadening and loss of the window. The peak velocity measured was 25.5 cm/sec.

4.4.d Flow Disturbance and Occlusion

The images of the following volunteer are included to demonstrate marked flow disruption, almost complete occlusion of the maxillary artery and strong bruit formations.

Figure 22.

Spectral Waveform - Rest State

The initial peak velocity was 43.9 cm/sec. The spectral waveform is sharp and normal in appearance.

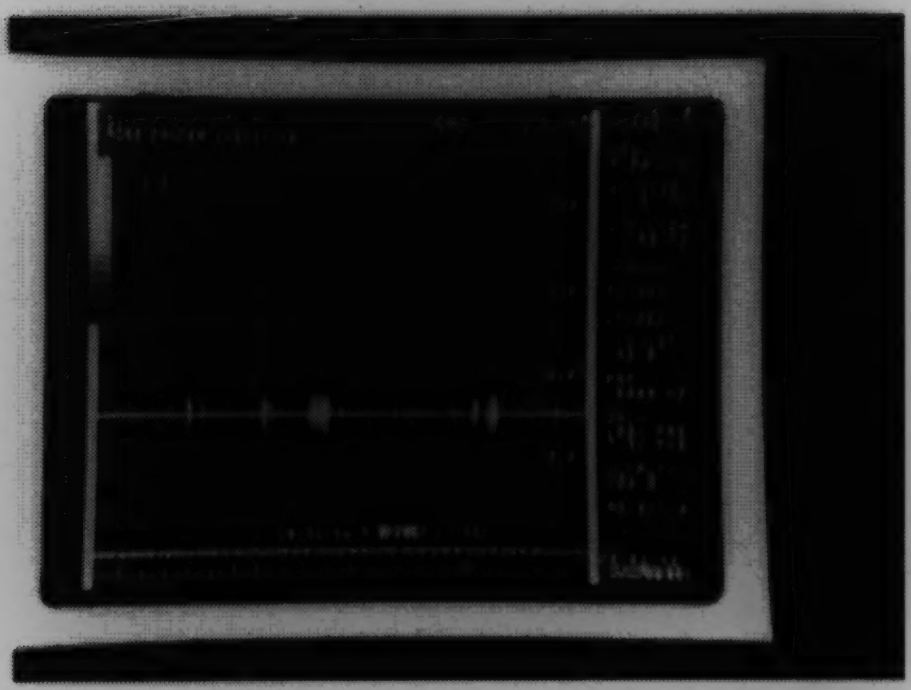


Figure 23.
Spectral Waveform - Clench State
Occlusion Present with Strong Bruits

Following 60 seconds of experimental clenching, the flow for this patient became very disturbed with signs of turbulence formation and prominent bruits. There were

noted intermittent bursts of flow measured at a velocity of 21.6 cm/sec but overall this patient demonstrated signs of almost complete occlusion of the maxillary artery lumen on the left side.

From the doppler ultrasound waveforms, the peak velocities for the right and left maxillary arteries were recorded both during rest and then during clenching.

The clenching velocities demonstrated a trend of diminished peak velocity following an initial increase in peak velocity suggestive of developing stenosis during external mechanical compression due to contracting hypertrophied lateral pterygoid muscle bellies. The increase in velocity was noted shortly following initiation of mandibular clenching. As the experimental mandibular clenching continued the peak velocities measured diminished to below rest peak velocity levels.

The diminished clench peak velocities are suggestive of increased luminal stenosis of the maxillary artery following the 60 second experimental time.

Table 4.

Peak Artery Velocities cm/sec: Symptomatic

Subject	Sex	Age	Flow Lt Rest	Flow Lt Clench	Flow Rt Rest	Flow Rt Clench
1	f	54	60	50	60	37
2	f	24	57	37	76	35
3	f	35	62	32	76	35
4	m	34	62	40	64	37
5	m	57	74	30	70	35
6	f	40	55	47	74	58
7	f	23	109	55	126	55
8	m	28	28	22	26	23
9	f	24	86	63	90	51
10	f	31	56	58	62	37
11	f	26	62	58	82	47
12	f	39	68	32	72	46
13	f	46	32	24	40	24
14	f	27	63	49	62	39
15	f	35	71	55	60	39
16	f	21	52	41	63	35
17	f	53	41	23	65	51
18	f	32	81	38	117	67
19	f	27	66	35	91	67
20	f	44	89	45	57	32
21	m	33	63	44	48	37
22	f	31	94	51	55	26
23	f	38	23	18	43	12
24	f	28	48	39	45	29
25	f	46	52	41	50	31
26	f	16	72	49	48	35
27	m	38	74	50	58	45
28	f	31	96	70	95	85
29	f	28	69	30	45	28
30	f	46	88	52	28	16
31	f	52	58	25	68	38
32	f	32	37	14	35	22
33	m	42	71	24	74	31
34	f	23	58	32	69	30
35	f	43	69	37	69	29
36	f	30	57	27	65	26
37	m	36	78	37	77	31
38	f	56	45	27	66	38
39	m	34	69	41	69	35
40	f	57	39	17	37	38
41	f	23	57	33	73	39
42	m	45	58	31	72	55
43	m	41	49	38	50	31
44	f	43	44	22	62	30
45	f	39	42	12	36	29
46	f	27	70	42	49	23
Mean ±SD		36 ±11	62 ±18	38 ±14	63 ±20	37 ±14

Table 5.

Peak Artery Velocities cm/sec: Normal

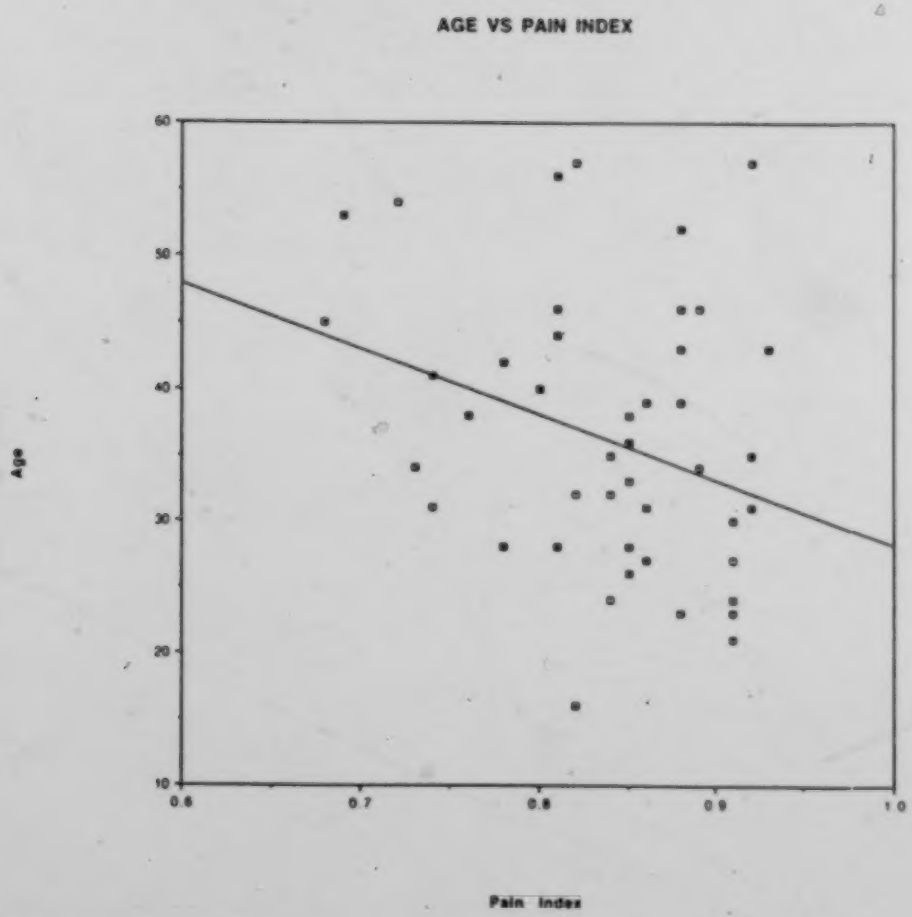
Subject	Sex	Age	Flow Lt Rest	Flow Lt Clench	Flow Rt Rest	Flow Rt Clench
1	m	15	67	67	74	73
2	m	17	61	60	59	59
3	m	18	68	69	65	64
4	f	18	74	74	59	58
5	m	20	54	52	49	48
Mean ±SD		18 ±2	61 ±9	61 ±9	60 ± 9	60 ± 9

From the numerical data observed in Tables 1 - 5, it was important to examine for relationships between the pain indices, muscle ratios, rest and clench peak velocities, and age.

The first relationship that was explored was that of age and pain indices. Using a linear correlation, at 95% confidence, a significant difference was noted (2-tailed significance = 0.04).

Age was plotted along the y-axis and the pain indices were plotted along the x-axis. There was an observed significance suggesting that the greater the age the more reduced was the pain indices. This suggests that there is a change in pain perception or awareness with increasing age.

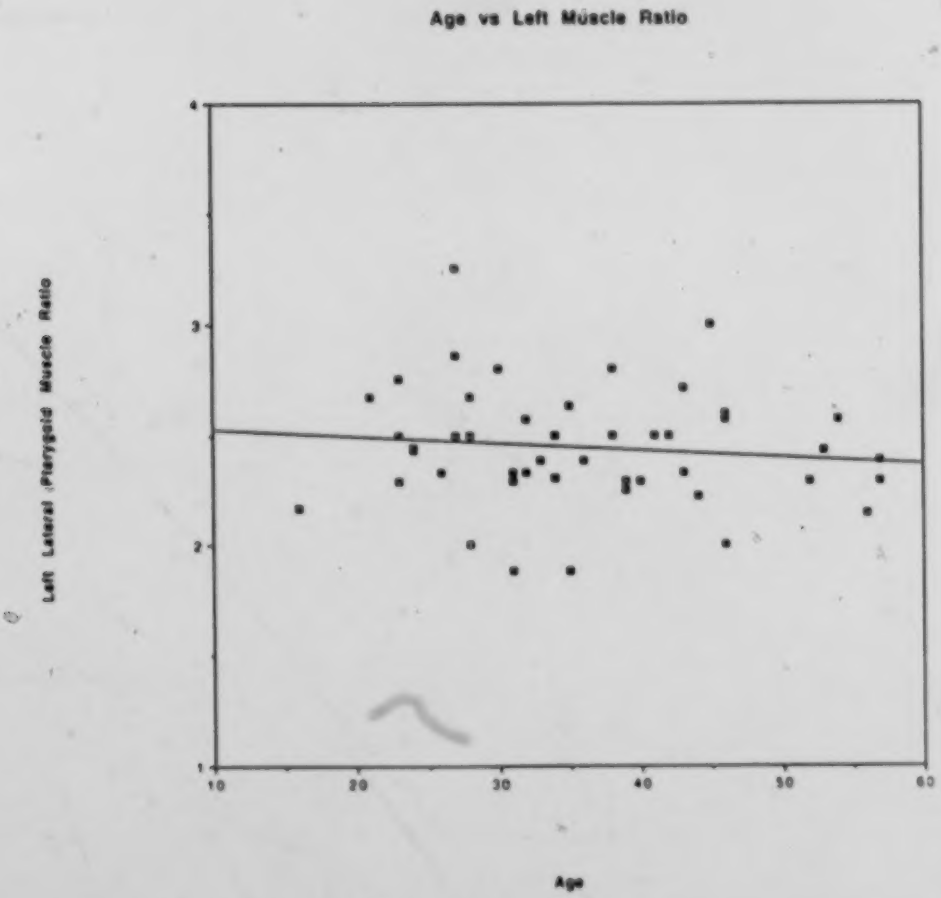
Graph 1.



The second relationship of interest was that of age and the lateral pterygoid muscle ratios. The relationship between the left lateral pterygoid muscle ratio and age

was examined first. A linear correlation was calculated and no correlation was found (p = 0.426)

Graph 2.



There is a evenly distributed trend noted in this graph suggesting random distribution and developed muscle ratio. There does not seem to be any correlation between the observed muscle ratios of the lateral pterygoid muscles with age. This suggests that the

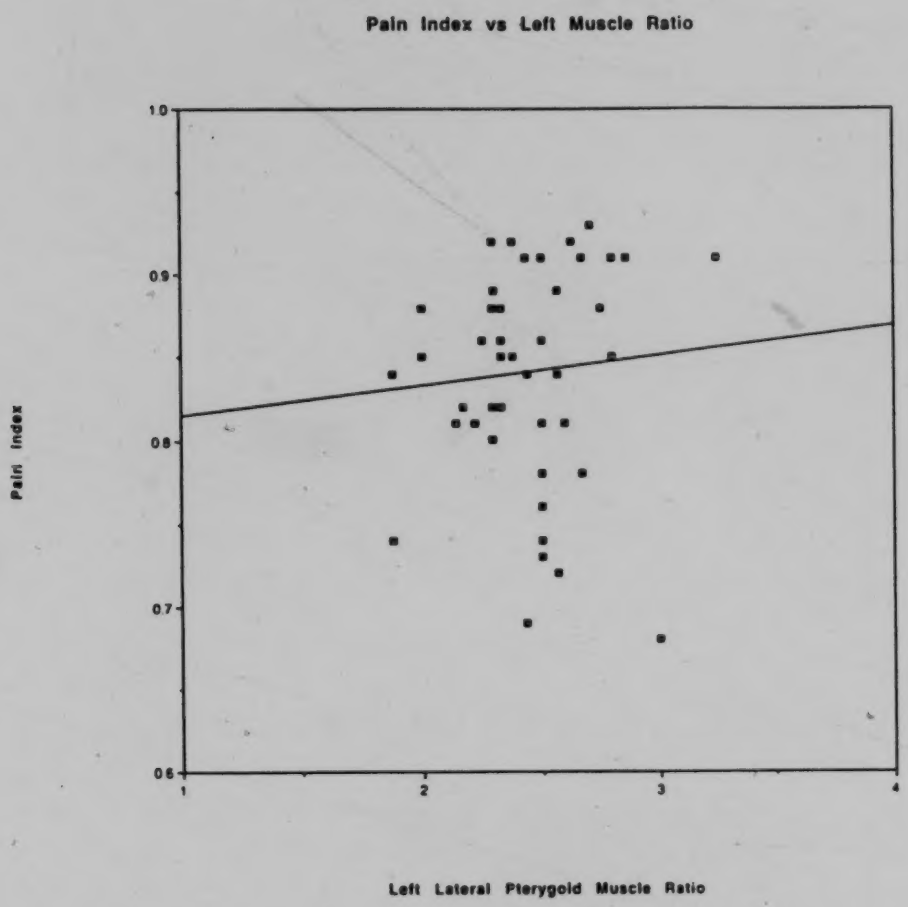
problem of hypertrophy of the lateral pterygoid muscles due to parafunction might be more a factor of the length of time a person has been clenching rather than their chronological age.

Plotting the right lateral pterygoid muscle ratios against age demonstrated a similar random distribution of ratios at all ages of the experimental cohort. As this graph was almost a mirror image of the graph plotting the left muscle ratio against age, the graph was not included.

The third relationship of interest based upon the hypotheses for this study were the relationship between the pain indices and the lateral pterygoid muscle ratios.

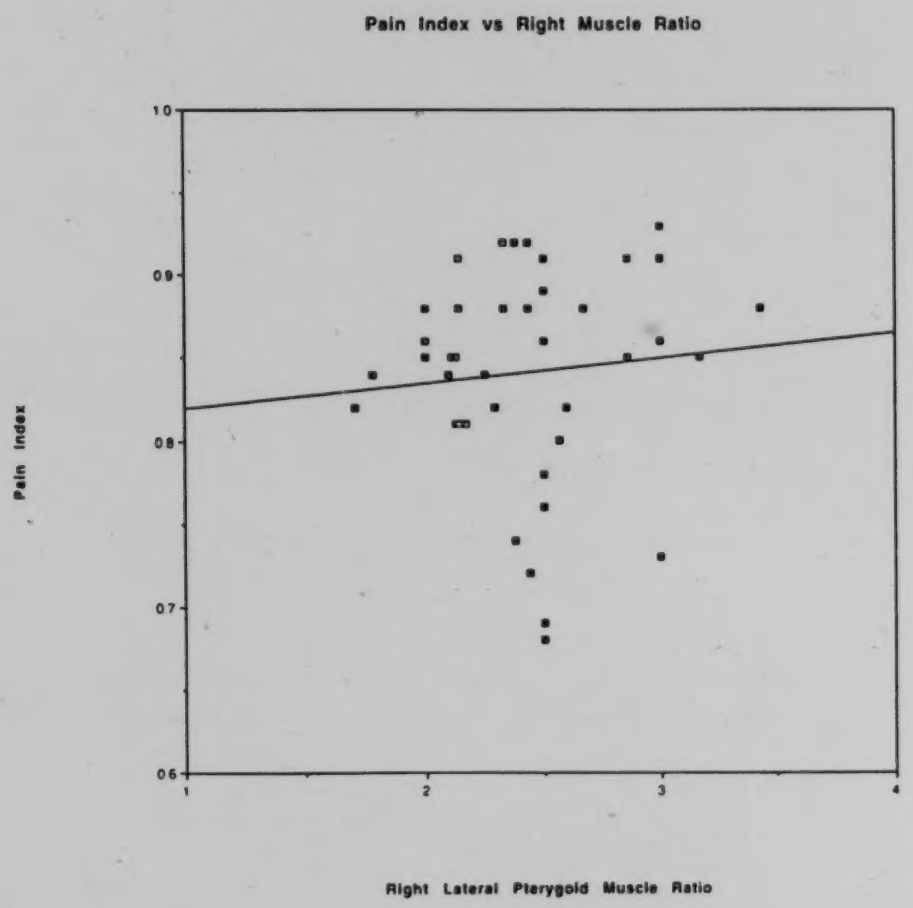
The first portion examined was pain indices against the left lateral pterygoid muscle ratio. The pain indices were plotted on the y-axis and the left lateral pterygoid muscle ratio was plotted along the x-axis. A linear correlation was performed and no significant correlation was observed ($p = 0.693$)

Graph 3.



From the extrapolated line there is a trend suggesting a relationship between increasing left lateral pterygoid muscle ratio and increasing pain indices. The majority of the volunteers seemed to fall within the range of 2.0-3.0 lateral pterygoid muscle ratio.

Graph 4.



The right muscle ratio plotted against the pain indices suggested a similar developing trend. A linear correlation test was calculated and no correlation was found for these measurements (p = 0.603).

Based upon my observations, and the suggested trend from the data, it is assumed that the greater the muscle hypertrophy, the greater the potential for producing pain and dysfunction via mechanical compression upon the maxillary artery.

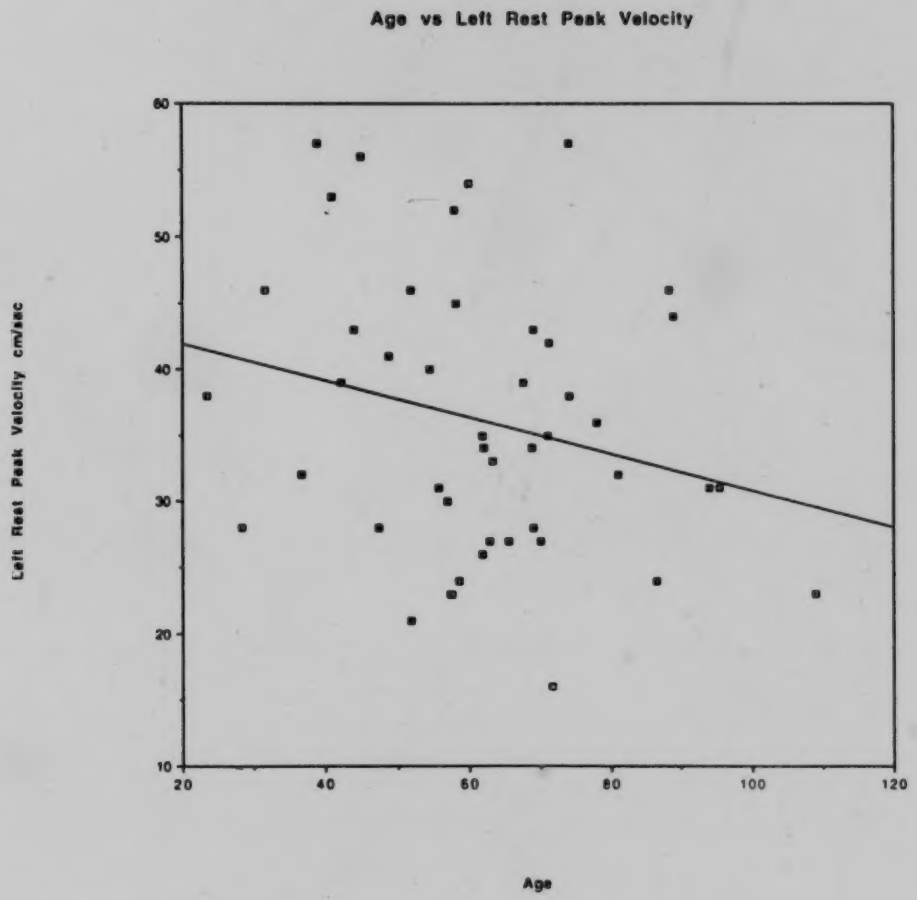
The overall test of the hypothesis for this particular study was the change in peak arterial velocity of the maxillary arteries during the rest phase without the lateral pterygoid muscles contracting and during contraction (mandibular clenching). The data for the rest and clench velocities were plotted against age to observe any possible relationships.

The first examination was plotting the maxillary left artery peak velocity during rest on the y-axis against age plotted on the x-axis. The standard error for the left peak velocity was 2.7. A linear correlation test demonstrated no significant correlation ($p = 0.109$). The plotted data suggests a trend of diminishing rest peak velocity within the maxillary artery with increasing age. The relationship could not be proven to be significant.

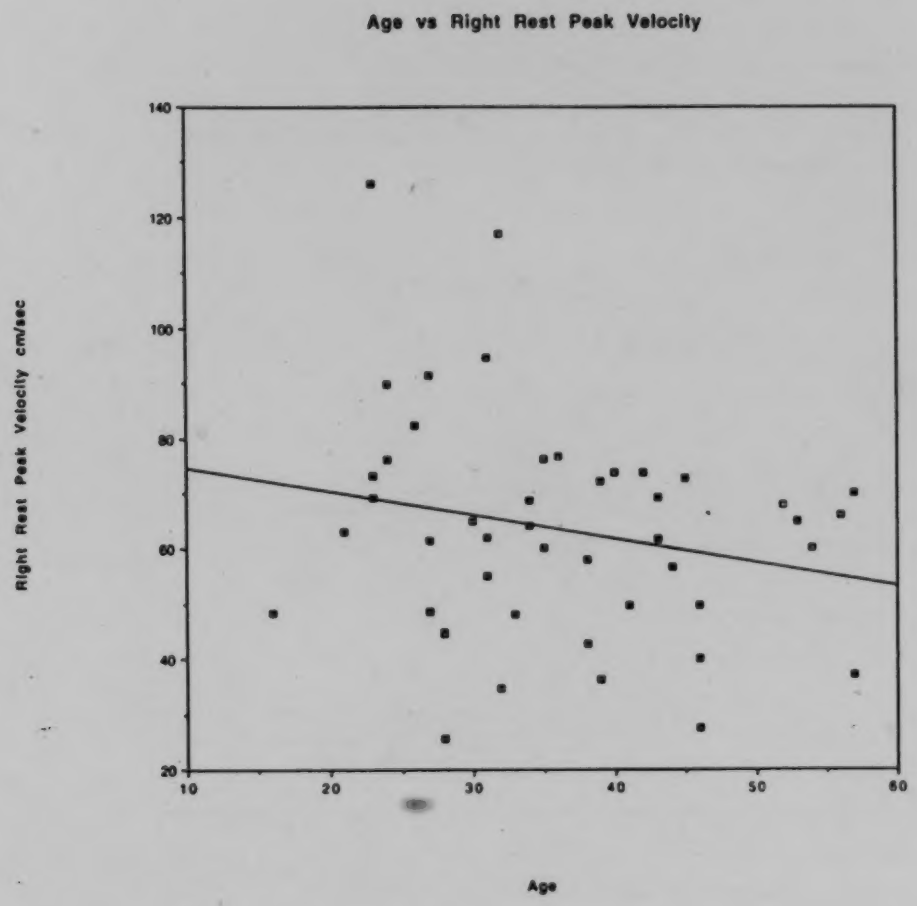
A possible explanation for such an observation might include decreased arterial flexibility in later years

and increase in arterial lumen plaque from atherosclerosis.

Graph 5.



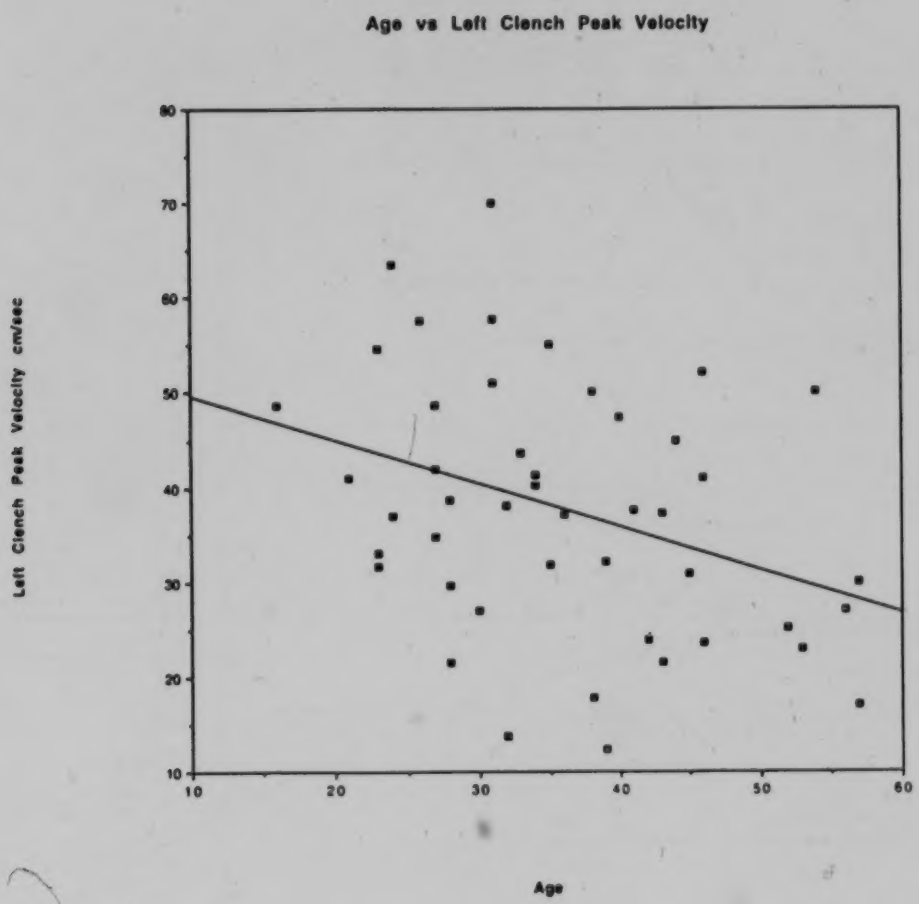
Graph 6.



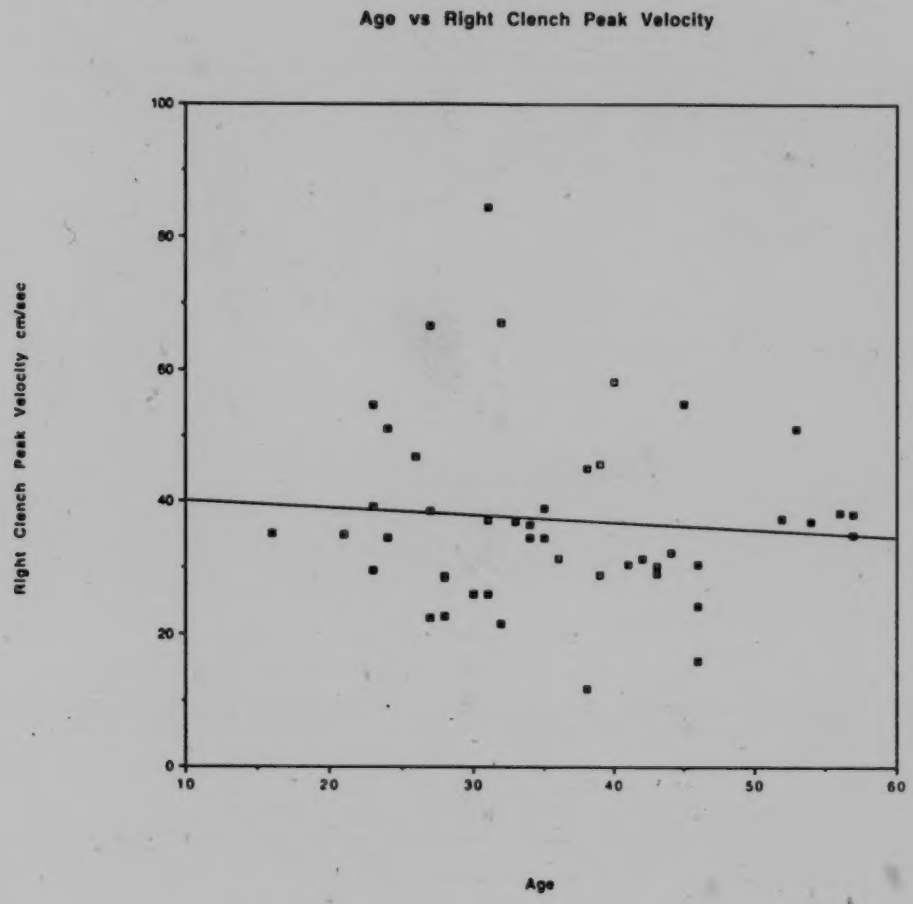
In this particular relationship, the right maxillary artery peak rest velocity was plotted against age. The standard error was calculated to be 3.0. A linear correlation was found to be non-significant ($p = 0.139$) This particular plot demonstrated a similar trend that was observed with the left maxillary artery peak rest velocity.

There appeared to be decreased rest peak velocities with increased age although the data could not be proven to be significant.

Graph 7.



Graph 8.



The previous two graphs (7&8) plotted left and right maxillary artery peak clench velocity. Both plots suggest a trend of decreased peak velocity with increasing age. The data for age and left clench flow were analyzed with a linear correlation and it was found to be significant ($p = 0.015$) The right clench flow and age data were similarly examined with a linear

correlation test and the measurements were found to be non-significant ($p = 0.575$). In spite of the lack of a significant correlation for the right clench flow and age, there is strong suggestion of a trend developing that is similar to the significance found with the left clench flow and age.

An age matched subgroup of symptomatic volunteers (N = 7, Age mean 22 $\pm$2) was compared with the normal group of volunteers to test for any correlation between rest and clench peak flow velocities. A significant correlation was noted between the normal volunteers who demonstrated only minimal change in peak flow velocity between the resting state and during clenching and the symptomatic group who demonstrated significant change in flow peak velocity (left clench velocity $p = 0.002$ and right clench velocity $p = 0.001$).

The noted significance in these small age matched subgroups suggests that there is a correlation between rest and clench peak flow velocities between the normal population and a symptomatic population.

The collected data from this investigation suggests a trend of lateral pterygoid muscle hypertrophy within a selected population of headache and facial pain sufferers. This hypertrophy was noted to affect peak

flow velocities of blood within the maxillary arterial system via mechanical compression of the arterial body. The mechanical compression was identified by the reduction of luminal diameter of the maxillary artery during mandibular clenching. Taking into consideration the error factor with utilizing ultrasound measurement, there is some question of validity of these measurements. If one considers the ratios and not the actual measurements, then the measurements demonstrate trends that suggest a possible relationship between the lateral pterygoid muscle bellies and the maxillary artery.

Increasing age appears to demonstrate a trend in headache and facial pain and was thought to possibly be related to stress handling capabilities and generation of hypertrophy of the lateral pterygoid muscles over time. The gender issue is consistent with reports within the literature of females suffering significantly more than males and even is this selected population of volunteers, the females out numbered the males approximately 4:1. The normal volunteer numbers also supported this ratio in that the males outnumbered the females by a 4:1 ratio. Estimates within the literature suggest ratios as high as 10:1 female to male.

A possible explanation for this is that this particular problem appears to have a strong psychosocial component to it. It is thought that perhaps males, vent frustration more openly and physically rather than by inward mechanisms such as clenching of their teeth. Females, on the other hand, appear to internalize their stress and vent it out more verbally and through nocturnal clenching (10,100,101).

The generation of headache is important in this study. The literature reports that these headaches are consistent with muscle contraction but the trends observed in this study suggests that there is more involved than can be explained by mechanically induced ischemia and fatigue.

The mechanical compression is not continuous but occurs intermittently throughout the day and during the sleep hours. The sleep hours are more important that the daytime clenching as there are powerful proprioceptive reflexes that exist within the teeth and periodontal ligaments that limit the ability to maintain clenching. A variant to this is the complete denture wearer. This persons do not have either teeth or periodontal ligaments to provide proprioceptive protection from overloading of the muscles and jaws in generating muscle pain.

Anecdotally, from my person experience, edentulous persons do suffer from these types of problems but their numbers are significantly lower than what is seen in the dentated population. A possible explanation for this might be decreased range of mandibular movements seen in the edentulous population due to accommodation within the masticatory system to decreased functional vertical dimension of occlusion. The inability to obtain solid clenching and bracing of the mandible with movable denture bases might also be a factor. Those persons who exhibit signs of MPD are those who exhibit gross loss of vertical dimension or occlusion and those who have been involved in trauma.

CHAPTER 5.

CONCLUSIONS

The purpose of this study was to investigate the maxillary artery and the lateral pterygoid muscles within the infratemporal fossa and to determine if hypertrophy of the upper and lower bellies of the lateral pterygoid muscles cause mechanical stenosis of the maxillary artery. If stenosis was generated, did it result in flow disturbances that manifested as pain and dysfunction within the craniomandibular complex.

In this study 51 volunteers were examined clinically and were imaged both with magnetic resonance imaging, ultrasound and doppler ultrasound. Upon examination and imaging, all volunteers demonstrated the maxillary artery arising medial to the temporomandibular neck. As the artery progressed through the infratemporal fossa, it passed medial to the lower belly of the lateral pterygoid muscle. Normal muscle mass as determined in the normal population demonstrated ratios of lower to upper belly lateral pterygoid mass of approximately 1.5:1 $\pm$ .2. The symptomatic population of this study exhibited mass ratios as high as 3.43:1. It appeared that with increasing mass ratios, there was a trend with increased pain indices and increased level of flow disturbances.

Pain indices and age were found to be statistically significant. Increasing age demonstrated decreasing pain indices.

Examination of the gender and age differences demonstrated consistency with what has been reported within the literature. The condition has a definite predilection for females in the second to fourth decade. The reported ratios of gender differences are usually in the order of 10:1. In this study, the ratio was 4:1.

Comparison of rest and clench values between the genders was important as both populations demonstrated diminished vascular peak velocities and flow disturbances following a period of increased flow peak velocities consistent with stenosis.

The number of normal volunteers (non-symptomatic) was low and this was due to the difficulty in finding them in an older population. The volunteers were all under 20 years of age. It appears as the carefree teenage years pass, the incidence of parafunctional clenching increases. This clenching has been reported due to increased life stress (100).

Colquitt (80) has reported sleep posture was a significant factor in clenching. Persons who tend to

sleep on their sides or abdomens tend to pivot their jaws laterally to the contralateral side that they lay on. This results in the mandible protruding asymmetrically. This protrusion brings the lower belly of the lateral pterygoid muscles into tone. As the person was usually clenching and not gnashing their teeth, the muscle action was isometric. Sleep posture in itself is not significant until stress factors are added into the equation. Persons who clench their teeth usually do so during rapid eye movement (REM) sleep patterns. The clenching has been suggested as a venting mechanism due to emotional stress. This stress is released through masticatory muscle activation during these sleep times.

The response to this isometric contracture is hypertrophy of the upper and lower belly of the lateral pterygoid muscles. The hypertrophy of the upper belly of the lateral pterygoid muscle occurs as the temporomandibular disc is postured out of the glenoid fossa with the orbiting condyle. As the action is primarily clenching, the upper belly of the lateral pterygoid muscle is performing an isometric contraction. The net result is hypertrophy of this muscle belly.

Hypertrophy of the two bellies of the lateral pterygoid muscles diminished the space within the infratemporal

fossa. During these isometric contractures, the first and second segment of the maxillary artery are in a position to become compromised by the hypertrophied muscle mass during clenching. This results in external compression of the maxillary artery and its tributaries.

The use of ultrasound imaging and doppler ultrasound imaging permitted observation in real time of flow through the maxillary artery as well as the bulking effect of the lateral pterygoid muscle during clenching. As the muscle bulked, the lumen of the observed segment of the maxillary artery was seen to consistently diminish.

Measurements of the lumen diameter during rest and during clenching demonstrated luminal narrowing consistent with stenosis. Release of the clenching allowed the lumen diameter to return to pre-clench dimensions in the majority of cases examined. Color flow doppler imaging demonstrated in real time the direction of flow as well as the presence of high velocity jets within the lumen. By manipulation of the color flow parameters, turbulence could be imaged by the presence of a specific color pattern (green).

A small subgroup of age matched symptomatic volunteers was tested against the normal volunteers and

statistically significant data was found suggesting that in the symptomatic group that there was reduction of the peak flow velocity within the maxillary artery during clenching.

The ultrasound also provided an audio signal of the flow. During clenching in some volunteers, audible bruits were heard that corresponded to the bruits seen on the spectral waveforms. In all the test subjects, when the clenching was started, there was a definite change in the audible pattern. The pitch increased corresponding to the increased jet velocity as the stenosis was building.

Doppler waveforms made of each volunteer demonstrated initially normal sharp spectral borders during rest with the teeth apart. There were well defined windows present. As clenching was initiated, spectral broadening was noted with increased peak velocity consistent with stenosis via external compression.

As the clenching continued, the increased blood peak velocity diminished to below initial resting levels due to diminished lumen diameter of the artery. As the clenching continued, the spectral waveform became consistent with disturbed flow. The spectral border became ragged, loss of the spectral window as flow

became delaminated and turbulence developed. In some patients, the disruption was so significant that almost complete occlusion developed. These patients were difficult to examine as their pain levels reached maximum very soon into the clenching cycle. Only the most stoic continued clenching for the entire 60 second clench cycle.

With the external compression of the maxillary artery, there is potential for pain generation by direct stimulation of the artery wall.(26) Pain can potentially also be generated by transient ischemia into the middle and accessory meningeal arteries. The accessory meningeal artery supplies blood for the gasserian ganglion. Ischemia to this structure is tantamount to negative stimulation that results in decreased flow velocity in the cerebral arterial system (31).

Stimulation via compression of the proximal portion of the middle meningeal artery and possibly torsional traction results in a response to neurons located within the trigeminal nucleus (33). These neurons are classified as nociceptive and the characteristics of their cerebrovascular activation is consistent with their role in mediation of vascular pain referring pain

to the peri-orbital and anterior temporal regions of the head.

This investigation took a selected population of craniomandibular pain sufferers and examined them regarding the relationship of the lateral pterygoid muscle mass due to hypertrophy and the effect during contraction upon the maxillary artery. Interesting trends were observed but due to design flaws in the volunteer group, statistically significant results were obtained but better design including age matched random selected volunteers from the population would yield more relevant statistics.

CHAPTER 6.

FUTURE STUDY

The trigeminal nerve transmits headache pain from blood vessels of the pia mater and the dura mater. Triggers for this pain are not well understood but are probably multiple with largely chemical mediators. Ischemia to the ganglion may be a factor.

The interesting questions that arise from this study concern the genesis of vascular headaches and the neural control alteration due to ischemia to pain receptor mechanisms. Is the vertebrobasilar system affected similarly by instability of the upper cervical spine vertebrae? Can this simple mechanism offer a sufficient explanation for the complex regional pain problems experienced by many patients under the guise of migraine or vascular headache?

It appears that clenching of the teeth parafunctionally is a strong etiological factor that results in transient ischemic attacks within the infratemporal fossa with resulting atypical facial and cranial pain. Clenching of the mandibular teeth against the maxillary teeth often occurs with the head placed into flexion thus exerting forces upon the ligaments of the upper cervical spine. This continual microtraumatic sprain force ultimately leads to laxity of this ligamental system. Laxity allows abnormal joint posture of the relationship

of the occipital condyles with the atlas and the axis. As the vertebral arteries are passing through this area, it is probable that they are being stretched and kinked resulting is flow abnormalities into the basilar system and posterior Circle of Willis. Flow abnormalities and aberrant innervation via the gasserian ganglion due to potential ischemic episodes may affect the central posterior circulation of the brain.

A random population of normal and pain suffering volunteers needs to be examined utilizing in addition to the criteria I investigated, a psychological profile to assess stress levels and the person's life coping skills. They need to be investigated in a sleep lab environment to determine preferred sleeping postures, level of sleep (REM vs deep delta patterns), and electromyographically monitoring the number of times clenching is induced during the usual sleep cycle.

This information in an age matched randomly selected population will provide data to support the hypothesis generated from this study and may prove statistically significant. In addition, more accurate measurement data is required relating to arterial lumen measurement. Angiography and possibly magnetic resonance angiography are suggested additional modalities that could be included within the experimental design.

A mechanical model of stenosis should be constructed to demonstrate the flow disturbances observed with doppler ultrasound. The same doppler ultrasound should be utilized with the mechanical model to demonstrate consistency with measurement.

An animal model is not suggested because at present, there is no animal that will clench their teeth, particularly during sleep. The animals seem not to be able to clench their teeth during waking hours due to innate proprioceptive protective reflexes.

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Appendix I

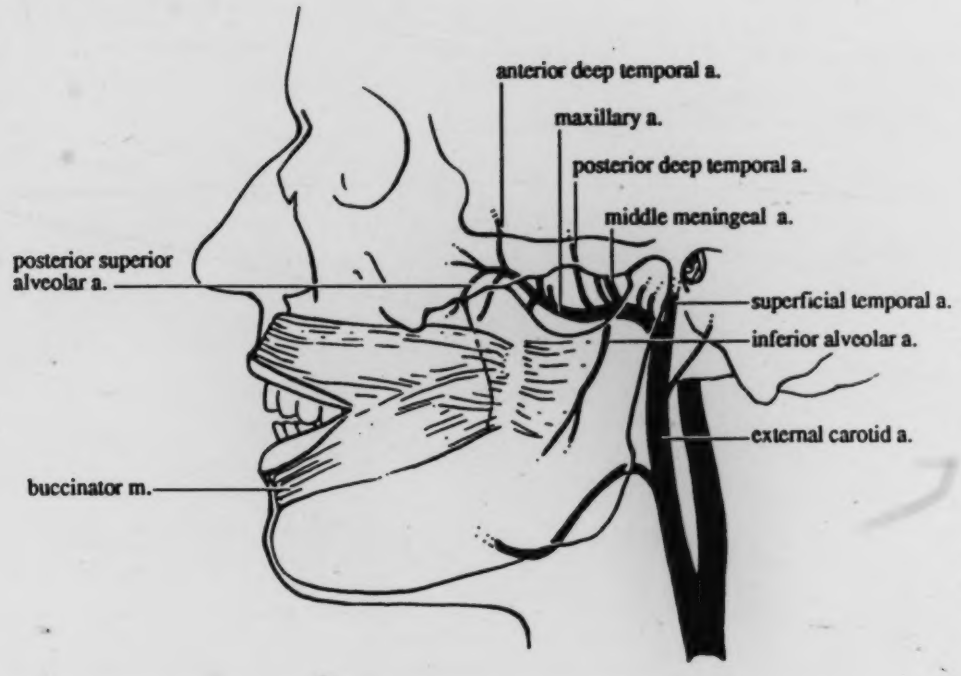
ANATOMY

Local Anatomy of the Maxillary Artery

The external carotid artery has two significant branches just as it passes the inferior border of the mandible at the level of the antegonial notch. Here arises the facial and the lingual artery.

The lingual artery before entering the tongue bifurcates to form the sublingual artery. This artery supplies the floor of the mouth and the mylohyoid muscle. This vascular supply is important as there is potential for anastomosis and collateral flow from this branch to the mylohyoid artery, a branch of the inferior alveolar artery which arises from the maxillary artery. This anastomosis may be responsible for the lack of bone pain expected with transient ischemia within the maxillary artery.

The facial artery supplies the superior constrictor muscle via the ascending palatine artery, the submental branch supplying the anterior belly of the digastric muscle and then ending as the superior and inferior labial arteries supplying the lips.



Orientation of maxillary artery on lateral view of skull.

Figure 24.

Arising superior to the facial artery is the occipital artery supplying the sternocleidomastoid muscles and produces a descending branch down into the muscles of the posterior neck often anastomosing with the deep cervical artery. There is a small meningeal branch that enters the skull through the hypoglossal canal and the jugular foramen and also an auricular branch to the auricle and a mastoid branch supplying the mastoid air cells and dura mater. There are terminal occipital branches that anastomose with the posterior auricular and superficial temporal arteries.

The posterior auricular artery arises just superior to the posterior belly of the digastric muscle dividing into three branches: the stylomastoid artery, the auricular branch and the occipital branch.

The ascending pharyngeal artery, a slender vessel supplies via the pharyngeal branches the pharynx, palatine tonsil and the auditory tube. Small meningeal branches enter the cranium via the hypoglossal canal and jugular foramen (posterior meningeal artery). The ascending pharyngeal artery terminates as the inferior tympanic artery.

The internal maxillary artery arises from the external carotid artery posterior and deep to the mandible at the level of the coronoid notch of the mandible. It is this artery that is of interest in this current study. After

arising from the external carotid artery, it then courses anteriorly and superiorly to cross medial to the mandible at the level of the condylar neck. The artery passes through the parotid gland entering the infratemporal fossa. The maxillary artery has numerous branches that supply most of the deep structures of the face. These structures include all of the muscles of mastication as well as all the facial bones and teeth. The maxillary artery has been described as being in three segments (figure 24).

The first segment contains the inferior alveolar artery which leaves the maxillary artery at 90 degrees inferiorly to enter the mandible at the lingula. This artery supplies the bone of the mandible and the teeth with its terminal branches being the mental artery and the incisive artery. There is a mylohyoid branch that exists the inferior alveolar artery just before it enters the mandible that supplies the floor of the mouth.

The first segment of the maxillary artery also gives rise to the deep auricular artery and the anterior tympanic artery which supply the external and middle ear. The deep auricular artery also passes through the external auditory canal cartilage to supply some of the retrodiscal tissues of the temporomandibular joint.

The middle meningeal artery exits the internal maxillary artery superiorly and passes usually in close approximation to the auriculotemporal nerve then passing through the foramen spinosum supplying the dura. Just anterior to the middle meningeal artery is the accessory meningeal artery. This artery enters through foramen ovale and supplies the middle cranial fossa meninges and the gasserian ganglion. It is this artery that is of most interest in this study due to the ramifications of its diminished flow will have on both sensory as well as sympathetic involvement from resulting from potential transient ischemia of the gasserian ganglion.

The second segment of the maxillary artery supplies the muscles of mastication and the buccinator muscle. The deep temporal arteries arise superiorly from the maxillary artery and enter into the temporalis muscle from the infratemporal fossa. The masseteric artery exits anteriorly coursing over the coronoid notch to supply the masseter muscle. This artery also supplies the anterior and deep aspects of the temporomandibular capsule. The pterygoid branches extend both superiorly and inferiorly from the maxillary artery to supply the medial and lateral pterygoid muscle. Just before leaving the infratemporal fossa, the buccal artery arises anteriorly to supply the buccinator muscle.

The third segment arise from the maxillary artery after is passes through the pterygomaxillary fissure. The branches include the posterior superior alveolar artery, the descending palatine artery, and the infraorbital artery supplying the maxilla. The vidian artery, the pterygopalatine artery and the sphenopalatine artery also arise from this segment of the maxillary artery and supply respective portions of the pharynx, eustachian tube and the nasal cavity.

At the location of the maxillary artery the external carotid artery ends. The terminal branches of the maxillary artery are the maxillary artery and the superficial temporal artery. The superficial temporal artery crosses the zygomatic arch just anterior to the external auditory canal. As the artery crosses the zygomatic arch the transverse facial artery arises from it anteriorly. Superior to this, the middle temporal artery arises to supply the temporalis muscle. The terminal branches of the superficial temporal artery divides into branches to supply the scalp.

There is strong collateral blood flow to most cranial structures except for the contents of the infratemporal fossa. Bilateral flow is equally affected as hypertrophy is seen to be bilateral.

Appendix II

CMI PAIN INDEX

NAME:_____

CRANIOMANDIBULAR INDEX FORM

DATE:_____

Mandibular Movement

- O Maximum Opening ____ ____
- O Passive Stretch ____ ____
- O Restriction (per examiner)
- O Pain on Stretch Opening
- O Jerky Opening
- O "S" Curve Deviation
- O Lateral Deviation
- O Pain on Protrusion
- O Limitation on Protrusion ____ ____
- O Pain on Laterotrusion (Right)
- O Limitation on Laterotrusion (Right) ____ ____
- O Pain on Laterotrusion (Left)
- O Limitation on Laterotrusion (Left) ____ ____
- O Clinically is/can Lock Open ___R___L
- O Clinically is/can Lock Closed ___R___L
- O Rigidity on Manipulation

TMJ Noise

R L

- O O Reciprocal Click
- O O Popping (Audible)
- O O Reproducible Opening Click
- O O Reproducible Closing Click
- O O Non-Reproducible Opening Click
- O O Non-Reproducible Closing Click
- O O Fine Crepitus
- O O Coarse Crepitus

Palpation

R L **TMJ Sites**

- O O TMJ Lateral Capsule
- O O TMJ Posterior Capsule
- O O TMJ Superior Capsule

Extraoral Muscle Sites

- O O Ant Temporalis
- O O Mid Temporalis
- O O Post Temporalis
- O O Deep Masseter
- O O Ant Masseter
- O O Inf Masseter
- O O Post Digastric
- O O Med Pterygoid
- O O Vertex

Neck Muscle Sites

- O O Sup SCM
- O O Mid SCM
- O O Inf SCM
- O O Trapezius Insertion
- O O Upper Trapezius
- O O Splenius Capitus

Intraoral Muscle Sites

- O O Lateral Pterygoid Site
- O O Temp Insertion
- O O Med Pterygoid (Sublingual)

DATE:_____

Mandibular Movement

- O Maximum Opening ____ ____
- O Passive Stretch ____ ____
- O Restriction (Per Examiner)
- O Pain on Stretch Opening
- O Jerky Opening
- O "S" Curve Deviation
- O Lateral Deviation
- O Pain on Protrusion
- O Limitation of Protrusion ____ ____
- O Pain on Laterotrusion (Right)
- O Limitation of Laterotrusion (Right) ____ ____
- O Pain on Laterotrusion (Left)
- O Limitation of Laterotrusion (Left) ____ ____
- O Clinically is/can Lock Open ___R___L
- O Clinically is/can Lock Closed ___R___L
- O Rigidity on Manipulation

TMJ Noise

R L

- O O Reciprocal Click
- O O Popping (Audible)
- O O Reproducible Opening Click
- O O Reproducible Closing Click
- O O Non-Reproducible Opening Click
- O O Non-Reproducible Closing Click
- O O Fine Crepitus
- O O Coarse Crepitus

Palpation

R L **TMJ Sites**

- O O TMJ Lateral Capsule
- O O TMJ Posterior Capsule
- O O TMJ Superior Capsule

Extraoral Muscle Sites

- O O Ant Temporalis
- O O Mid Temporalis
- O O Post Temporalis
- O O Deep Masseter
- O O Ant Masseter
- O O Inf Masseter
- O O Post Digastric
- O O Med Pterygoid
- O O Vertex

Neck Muscle Sites

- O O Sup SCM
- O O Mid SCM
- O O Inf SCM
- O O Trapezius Insertion
- O O Upper Trapezius
- O O Splenius Capitus

Intraoral Muscle Sites

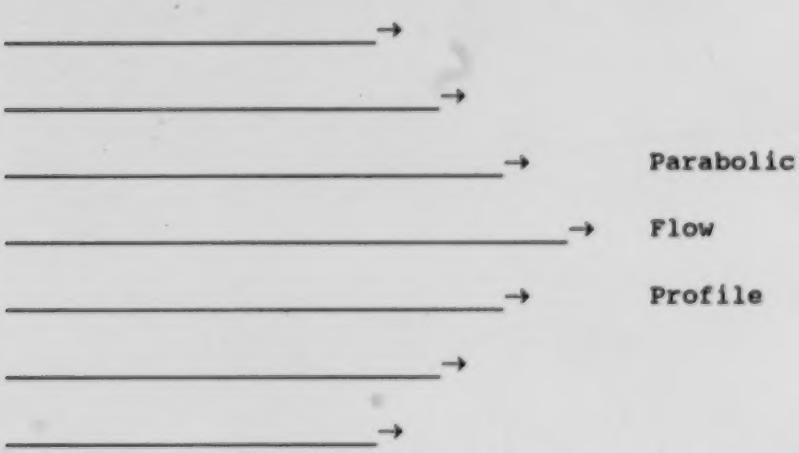
- O O Lateral Pterygoid
- O O Temp Insertion
- O O Med Pterygoid (Sublingual)

Appendix III

SPECTRAL ANALYSIS

III.1 SPECTRAL ANALYSIS

Blood flow in the healthy hemodynamic human system is a pulsatile steady flow. The flow in health is laminar. This implies that the stream lines run parallel to each other and to the vessel walls. The maximum flow speed occurs at the center of the vessel where the blood cell particles meet almost zero resistance and at the periphery the flow is significantly decreased due to friction.



Successive cylindrical layers (laminae) of fluid slide on each other with relative motion. There is a pressure difference at the ends of the tube that overcomes the viscous resistance to this relative motion, maintaining laminar flow. (96,97,98)

Situations creating stenosis occur primarily with disease that alters the laminar flow profile. These alterations include disturbed flow and turbulent flow.
Disturbed flow occurs in the region of stenosis within a vessel or at a bifurcation with the flow occurring in a forward direction. Turbulent flow is a flow pattern that is usually random and often chaotic yet maintains a net forward flow pattern.

Stenosis is any narrowing of a vessel lumen diameter either due to plaques on the interior of the vessel or external compression. It is external compression that is of interest in this study.

III.2 Appearance of Waveform:

The normal direction of systolic flow conventionally is displayed on the top half of the baseline and retrograde flow is represented on the bottom of the baseline (figure 16). Normal laminar flow demonstrates a bright concentration displayed near the outer edge of the waveform or envelope indicating most of the blood cells are moving at maximal velocity. There exists a dark area in the center of the waveform that is referred to as the window and it signifies a lack of low velocity motion of blood cells during the systole phase. The shape of the waveform

envelope is significant. A well defined envelope with sharp edges are typically seen with normal laminar flow.

As flow is disturbed and turbulence increases, the envelope becomes ragged and less distinct.

During peak systole, the distance between the outer border at the peak of the waveform and the upper border of the window is referred to as the spectral width. The spectral width can be fairly narrow with a prominent window in a normal laminar waveform (figure 16), or it can be spread out and widened with filling in or absence of the window when the flow becomes disturbed or turbulent. This is known as spectral broadening (figure 17).

As flow disturbances increase and turbulence develops and increases, eddying and severe disturbance may show a very bright concentration of flow at the baseline at peak systole and a poorly defined envelope with increased systolic velocity during the postsystolic/diastolic phase. This is known as a bruit or a gruff and may have the appearance of a seagull (figure 23) (96).

III.3 Arterial Stenosis:

Proximal to the stenosis the waveform exhibits a normal or slightly blunted appearance. The separation between the first and second components (dicrotic notch) may disappear due to vascular elastic changes along the stenosed area (figure 13). The window may show some flow scattering throughout.

Across the stenosis there is increased peak systolic velocity that increases proportionally with the flow velocity; the higher readings correspond with luminal reduction and this is referred to as a jet effect. The envelope begins to break up. The velocity will usually increase directly with the degree of stenosis. The window may also begin to disappear as spectral broadening increases. There is an increase in the audible pitch of doppler signal heard.

Baker 1978 (23) reported that cerebrovascular flow remains nearly constant until a hemodynamically significant stenosis (60 -70% luminal reduction) occurs.

Immediately distal to the stenotic area, disturbed or turbulent flow may be found ranging from strained burbling to a high pitched hissing doppler signal. At this point, the spectral waveform has a very ragged appearance with an

indistinct envelope, with widely distributed frequencies and a completely absent window. The severity of the stenosis can be judged from the level of envelope destruction and frequency distribution. Turbulence and eddy formation will cause an increase in the higher energy intensities at the baseline.(96,97)

In severe cases, a completely disrupted waveform with apparent velocity reductions at systole will result (figure 23).

Signals obtained more distally from the stenosis and away from the turbulent region may assume the appearance of a reduced waveform with even intensities and broadening throughout with disturbed envelope edges and occasionally no visible flow components. The peak velocity may be markedly diminished compared with the peak of the waveform seen directly at the level of the stenosis. The peak frequency may be markedly rounded. Severely diminished flow may become almost flat in appearance. Simultaneous forward and reversed flow occurs due to swirling of blood, and the spectral outline becomes irregular because of complete disruption of the laminar flow pattern. (96)

Total occlusion results in a sharp thudding or thumping sound which may be heard in the patent portion of the vessel proximal to the occlusion.

As flow is disturbed or becomes turbulent, greater variations in velocities of various portions of the flowing blood particles produce a greater range of doppler shift frequencies. Upstream (proximal) to a severe stenosis, lower mean velocities are seen and higher pulsatility ratios are observed. Tertiary effects are likely to occur within the collateral channels that bypass the obstructed region. Onset of turbulence is predicted by Reynold's Number. (97)

$$\text{Reynold's Number} = \frac{\text{avg. flow speed} \times \text{tube diameter} \times \text{density}}{\text{viscosity}}$$

In a rigid tube system, if the Reynold's Number exceeds 2000 then the flow is turbulent.

Yongchareon and Young 1979 (24) found that although there were variations reported in the literature for the critical Reynold's Number for the development of turbulence, their results clearly showed that turbulence would develop at relatively small Reynold's Numbers and the critical Reynold's Number decreased as the severity of stenosis increased. They recognized that pulsatile flow produced

more complex calculation problems. The critical Reynold's Number for the development of turbulence in pulsatile flow through a stenotic obstruction depended on numerous factors including stenosis shape and size and the nature of the base flow waveform.

They found that turbulence will develop at Reynold's Numbers well below the critical value for an unobstructed tube. In their study, they found that the critical Reynold's number was reduced as the stenosis shape became more abrupt. They also found that the critical Reynold's number varied with frequency parameters with the flow first becoming less stable and then more stable as the frequency parameter was increased. The axial location (critical length) at which turbulence was first observed was a function of both stenosis shape and frequency parameter. with the shortest length observed for the orifice. The critical length tended to decrease as the frequency parameter increased. They finally concluded that as the Reynold's Number was increased beyond the critical value that the location of the most intense turbulent fluctuations moved upstream. They noted that the turbulence induced effects resulted in post-stenotic dilations.

The average flow speed within a stenosis must be greater that the proximal and distal area so that volume flow can be constant throughout the tube. This is supported by the

Continuity Rule that states volume flow must be consistent for the three regions at a stenosis, proximal, at the stenosis and distal. This is because fluid is neither created nor destroyed as it flows through the tube. The experimental data supports this rule as the patient starts their clenching cycle and external compression affects the maxillary artery. There is an initial increase in flow speed but as the compression continues and the extent of the stenosis increases the velocity diminished significantly.

If the radius of the entire vessel is decreased as in vasoconstriction or in an extended area of stenosis the flow speed is decreased and if occlusion is approached flow speed is rapidly diminished. Spencer and Reid (1979) (21) suggested that as the diameter of a stenosis is reduced, the fluid volume is unaffected initially because the stenosis does not contribute substantially to the total vessel resistance. As the diameter continues to decrease the tube resistance increases, reducing the volume flow eventually to almost zero at occlusion. As the stenosis diameter decreases the flow speed initially increases to satisfy the continuity rule and reaches a maximum then starts diminishing to eventually zero due to occlusion of the vessel as the increasing flow resistance effect dominates.

APPENDIX IV

ETHICS APPROVAL

Office of the Dean
Faculty of Medicine

212 (0 WC Mackenzie Health Sciences Centre
Telephone (403) 492-6621
FAX (403) 492-7303

RESEARCH ETHICS BOARD

ETHICS APPROVAL FORM

Date: February 1991

Name(s) of Principal Investigator(s): Dr. Danny R. Kolotyluk

Department: Dentistry

Project Title: Comparison of Multiplanar Magnetic Resonance Imaging With Computed Tomography of the Human Temporomandibular Joint and Adjacent Structures.

The Research Ethics Board for Human Experimentation has reviewed the protocols involved in this project and has found them to be acceptable within the limitations of human experimentation.

Specific Comments:

Signed - Chairman of Research Ethics Board

for the Faculty of Medicine
University of Alberta

This approval is valid for one year.

\ethics\approve